A.M.A.

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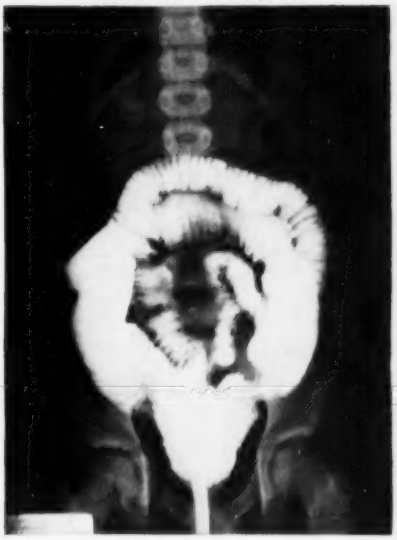

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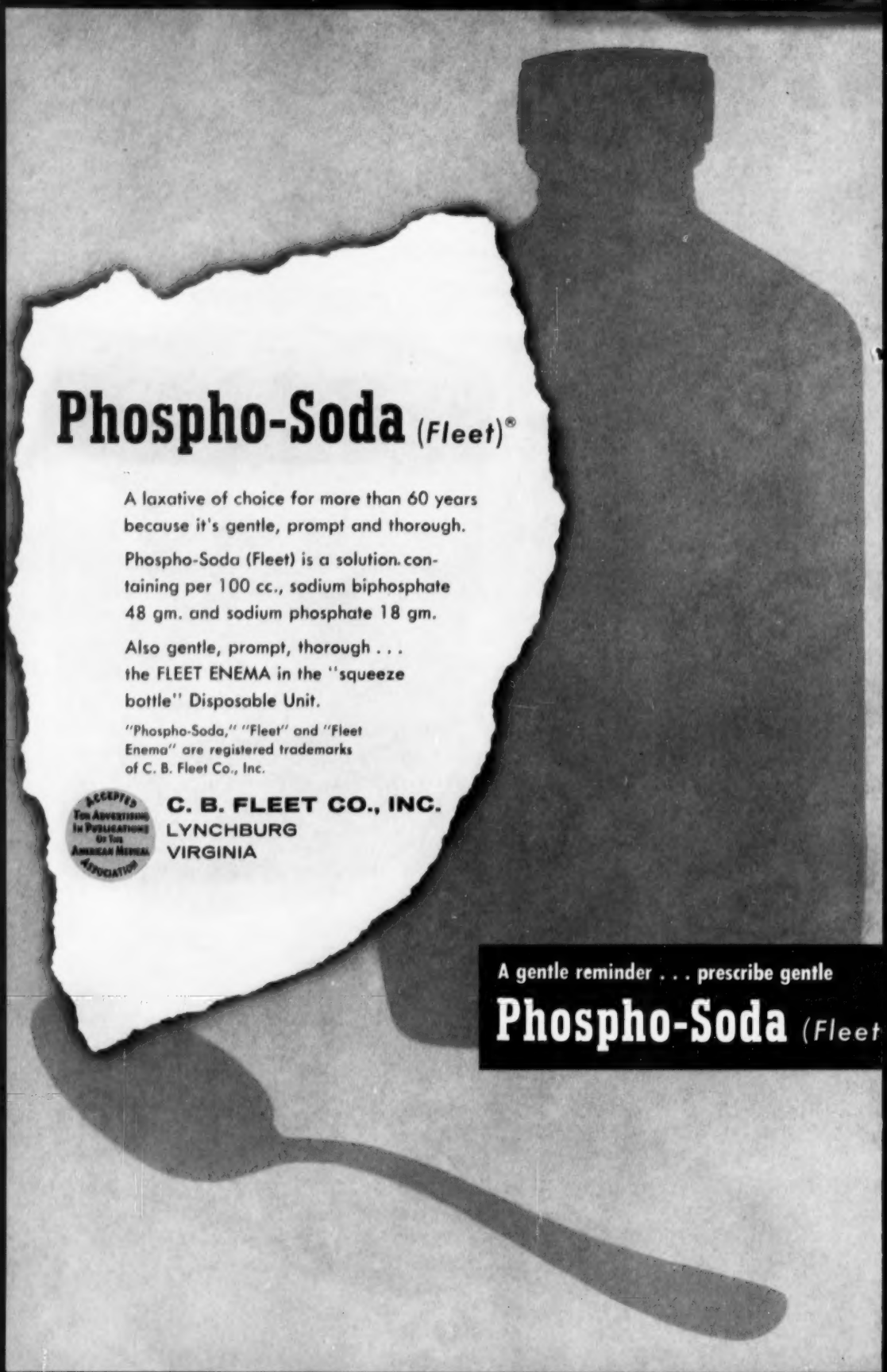
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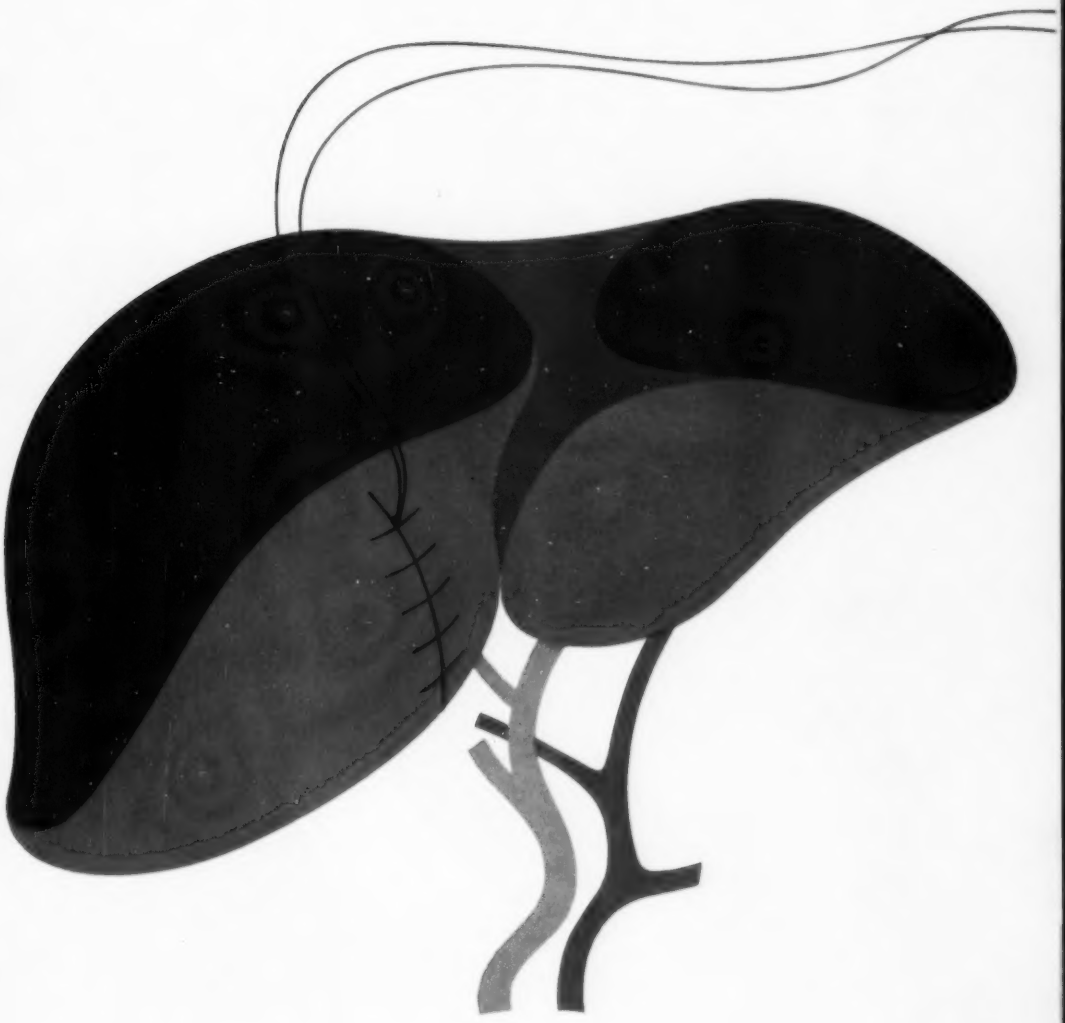

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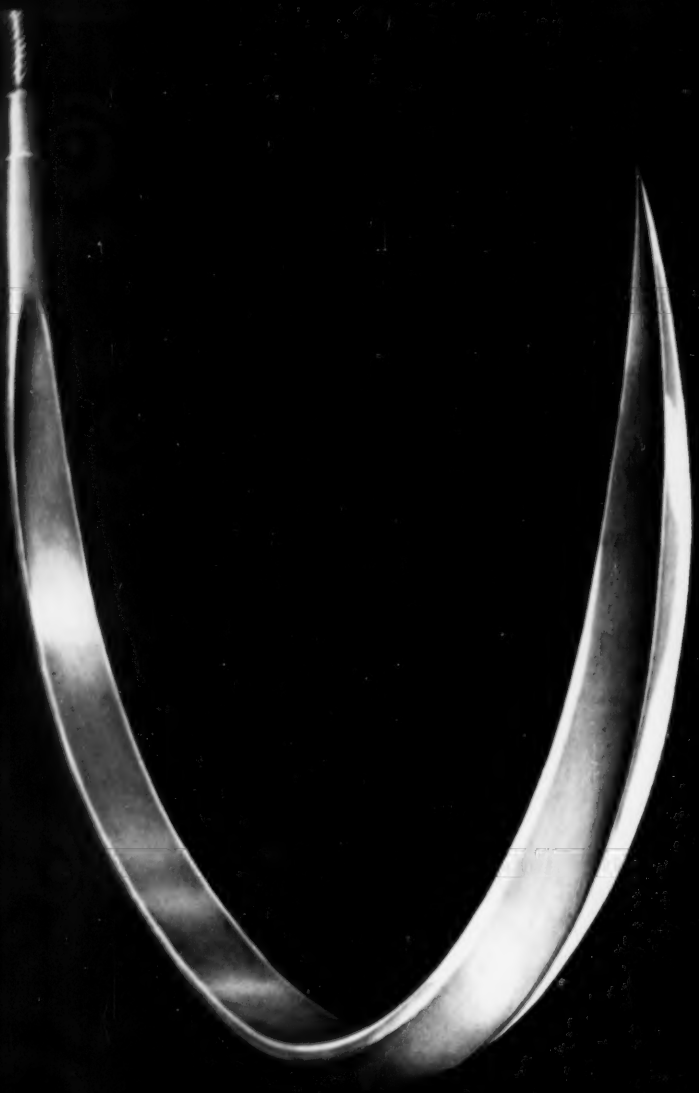

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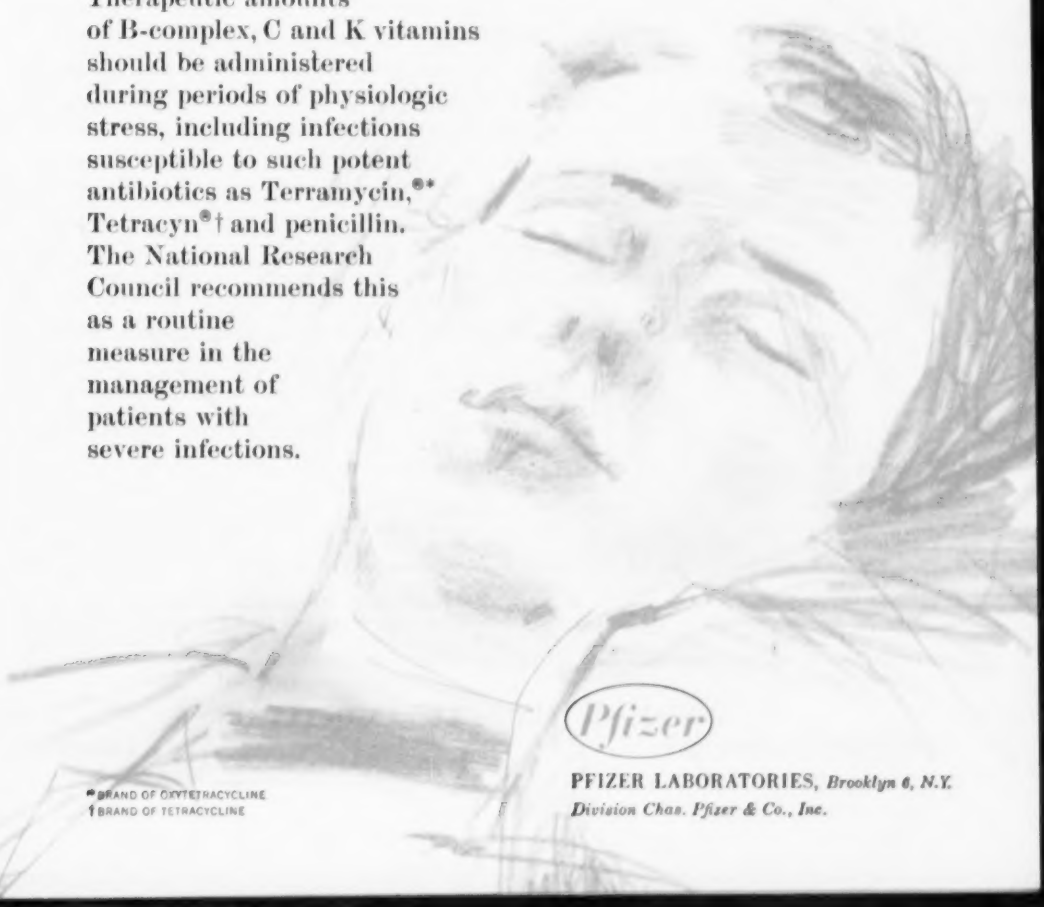

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Editorials

OPEN VS. NEEDLE BIOPSY IN THE EARLY DIAGNOSIS OF PROSTATIC CANCER

THE RESULTS of open perineal biopsy are compared in the present issue of A. M. A. Archives of Surgery [1] with those of perineal "punch" biopsy for the purpose of finding which is better for the early diagnosis of unsuspected prostatic carcinoma. This editorial is a word of caution against the indiscriminate application of these experimental results in clinical practice.

We know that about 20% of men over 55 years of age have prostatic carcinoma, since occult neoplasms have been repeatedly found at this rate in routine autopsies. Thus if a large enough biopsy (i. e., total prostatectomy) were done in all men over 55, we would find at least 20% positive specimens. The authors found that only 10.5% of their random extensive open biopsies were positive; in other words, the open method missed half the expected number of cases of cancer. In order to detect all the instances of cancer of the prostate in men over 55, routine radical prostatectomy would be necessary.

Now, a single "punch" biopsy of an unsuspected case would compare with an extensive open specimen in proportion to the amount of tissue removed, since both specimens are taken at random and the occult lesions could lie in any portion of the true prostate. To indicate that the "punch" biopsy was erroneously negative in as many as 15 of 21 cases subsequently proved to be positive (by open biopsy, itself 50% erroneous) distorts the fact that clinically the needle biopsy has a greater usefulness. Kaufman, Rosenthal, and Goodwin [2] found an accuracy of 61% for the needle biopsy (using multiple punctures) in relatively early (curable) prostatic carcinoma. Colby [3] found an over-all accuracy of 73% for needle biopsies. The accuracy was greater and approached 100% in advanced cases. They had better results because they had a palpable lesion to aim for. And this will apply for most of us in our clinical practice.

I would agree with the conclusion of Dr. Hudson and his co-workers that the punch biopsy is of greater value in proving suspected cases than for the detection of cancer at random. However, their conclusion that extensive open surgical biopsy is a better means of detecting early unsuspected cancer is subject to two criticisms. First, even such a major technique is only 50% accurate. And second, open biopsy has little clinical justification in unsuspected cases, since the authors cannot propose doing routine open perineal biopsies on all old men.

Frank Hinman Jr., M.D.

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ACUTE OBSTRUCTION OF THE SMALL INTESTINE

THE THERAPY of intestinal obstruction has become somewhat confused in the minds of many surgeons in recent years. The introduction of the long nasogastrointestinal tubes plus widespread use of antibiotics has produced a temporary improvement in the patients' condition that is often dangerously misleading to the clinician. Thus corrective surgery has been delayed to an extent that failure often results. In an attempt to clarify the situation as we see it at this time, I will set down some of the basic principles involved, with the hope that they will be helpful.

Paralytic ileus was common in the days when peritonitis was prevalent. Now it is infrequent and should not be confused with mechanical obstruction. There is always a physiologic ileus following laparotomy, the degree and duration of which is dependent on the amount of necessary manipulation of the small bowel. Even in extensive abdominal procedures, peristalsis returns to normal in about 48 to 72 hours. Since most of the intestinal gas is due to swallowed air, routine use of gastric suction prior to, during, and immediately after anesthesia has reduced the formerly seen postoperative distention with subsequent "gas pains" to a minimum. True paralytic ileus can still occur if the peritoneal cavity has been soiled or there has been excessive trauma from the freeing of extensive adhesions, or by the failure of routine use of gastric suction, which should be continued until the patient passes gas or peristalsis has returned to normal.

In paralytic ileus the patient may be uncomfortable and even restless but does not have the acute agonizing pain seen in mechanical obstruction. There is no "point" tenderness, although palpation will reveal a generalized soreness which is often sufficient to cause "guarding" by the abdominal muscles. Probably the most important use of the stethoscope by the surgeon is for auscultation of the abdomen. Early in his career he should learn to identify the sounds of normal peristalsis. In paralytic ileus there are no peristaltic sounds whatever if the stethoscope is used prior to vigorous palpation—the temporary change in position of contents in a gas- and fluid-filled loop of bowel by deep pressure may result in one "gurgle," and that is all. Such a mechanical sound should never be confused with the regular, continued peristaltic waves in the normal or obstructed bowel. The leucocyte count is not elevated in paralytic ileus in the absence of peritonitis or infection. The scout roentgen film of the abdomen shows a diffuse clouding, with all identifiable loops of small bowel increased in size to the same extent.

Paralytic ileus should be treated conservatively by gastric or long-tube suction, supportive intravenous therapy, and sedation. Surgery would serve only to further endanger the patient's life by prolonging the paralytic process. Ileostomy, so frequently done in former years, never served any useful purpose in paralytic ileus after repeated gastric aspiration by syringe was instituted, long before the advent of mechanical suction devices.

Mechanical obstruction of the small bowel is, at the earliest characteristic diagnostic signs and symptoms, a surgical emergency. Adhesive bands, internal or overlooked hernia, foreign bodies, torsion, or thrombosis are best treated by early operation. Patients appear in a state of dehydration, electrolyte imbalance, and with other signs of toxicity so that immediate corrective therapy may be advantageously instituted for a few hours prior to laparotomy. Prolonged conservative measures under these circumstances, on the basis of the obvious clinical improvement, is dangerous and avoidable.

Severe, almost unrelievable pain initiating mechanical obstruction is always present, and in patients admitted in the late stages of this disease information regarding the character of the onset can be obtained from the patient's family or physician. In strangulation obstruction or thrombosis, the leucocyte count is almost invariably elevated early. Even in fairly late cases before perforation of the bowel has taken place, the peristalsis is high-pitched and regular, and each wave is associated with increased pain. The scout roentgen film will show widely dilated loops of small bowel. The approximate location of the obstruction can frequently be ascertained by the observation of these films, when the gas pattern can be seen to end abruptly. Under no circumstances should a patient suspected of obstruction be given barium by mouth. Vomiting appears early in this disease, and the higher in the intestinal tract the stoppage, the earlier vomiting occurs.

Laparotomy should be done as soon as the patient's condition permits. Gastric suction is instituted as soon as the patient enters the hospital. Adequate anesthesia is imperative and should not be local infiltration in type. Owing to the dangers of blood pressure changes, spinal anesthesia is feared by many anesthesiologists in the older age group. Modern gas-oxygen-ether inhalation is usually the safest and is satisfactory. The incision should be liberal, usually longitudinal and paramedial on one side or the other. One must be prepared to meet a situation of bowel distention sufficient to necessitate a partial or, at times, an almost complete evisceration of the small bowel to get at the source of obstruction. The local procedures, done by dead reckoning, such as ileostomy, were seldom satisfactory, and they have been practically abandoned in modern times.

Often the question is raised as to what should be done when dealing with a severely ill patient with small bowel obstruction. The answer to this has too frequently been: Do the minimum procedure commensurate with hope of temporary relief. This, I am sure, is, on the whole, dangerous advice. Rarely is it necessary to curtail a well-planned operation that completely relieves the patient of obstruction. The improvement of the patient's condition on the operating table has long been recorded by our predecessors. Not only must one cut the constricting band, remove the gallstone in the ileum, or resect nonviable bowel, but one must consider other factors.

Although one may be content with releasing the obstruction in early cases, too frequently the patient is left with an enormous quantity of toxic small bowel contents which at certain stages of the disease will cause death. It is impossible to define the exact borderline under such circumstances when it is safe or not advisable to allow these obstructed contents to flow through the normal channels. With the release of obstruction, the blood supply, previously suppressed, soon allows normal absorption to take place. Also, the protective bacterial flora may have been changed into a more virulent one. We look back on useless fatalities in some of these cases and believe that many of them could have been avoided. This was recognized by our predecessors, and although their explanation of the cause of death might not meet the modern scientific concepts, patients still die if the stagnant material is left in situ.

Over half a century ago surgeons were not always able to differentiate between the effects of peritonitis, paralytic ileus, and many forms of intestinal obstruction. Thus the early literature is confused by the advocates of intraperitoneal or intraintestinal flushings with salt solution and the opposite school of thought demanding the minimum of trauma and therefore the least possible form of surgical intervention.

Although the latter viewpoint seems to have been generally abandoned, there has been a renewed interest in intestinal evacuation and, to some extent, intestinal flushing.

Having observed the bad effects of allowing these small bowel contents to remain for nature to cope with, I have long practiced and advocated the withdrawal of this material on the operating table. The late George H. Monks, more than half a century ago, made significant contributions regarding "intestinal locations" and followed this with further research concerning the emptying of the intestinal contents by gradually fluting the bowel over a rigid, jointed metal tube of large caliber. Later he added flushing of the bowel through two or three segmental enterostomy openings. Having assisted, many years ago, the late Charles Allen Porter, in a desperate situation of long-standing low small bowel obstruction, gradually to empty the intestinal content into a basin, I have been impressed by the importance of some such procedure. Although I am not convinced of the advantages of flushing the bowel with salt solution after it is emptied of most of its contents, I am sure that elimination of the major portion of this material is often of vital importance. Whatever modern gadget is used with suction on the table to accomplish this purpose is immaterial. Always one must depend on a capable, understanding assistant or two to gently and gradually allow one loop after another to empty its contents into the loop exteriorized and opened. It is amazing that such a procedure can be accomplished with minimum trauma and intraperitoneal soiling. The ease with which the emptied intestine can be replaced within the abdomen is in itself enough to recommend it. The improvement in the appearance of the bowel through its released blood supply further supports this thesis. I am sure that only in very early obstruction with minimal small bowel stasis should the contents be left undisturbed.

ARTHUR W. ALLEN, M.D.
Boston

Experimental Segmental Pericarditis

Its Bearing on Clinical Pericardiectomy

HARRY G. PARSONS, M.D.
and
EMILE HOLMAN, M.D., San Francisco

The wide diversity of opinion as to where and how much of the pericardium should be removed from the constricted heart in the surgical treatment of constrictive pericarditis has been the subject of much discussion and some controversy by many authors.* A preliminary report of certain significant experimental studies was presented to the Surgical Forum of the American College of Surgeons in 1950.[26]

Experiments have been many and varied.† In an excellent report, Isaacs, Carter, and Haller [23] presented highly important studies in which segmental constrictive pericarditis was produced by suturing the pericardium to the myocardium to form a pericardial pocket over the area of future constriction and introducing a copolymer vinyl acetate and vinyl chloride cast into the pocket to cause scarring. They presented a concept of the pathologic physiology of constrictive pericarditis supported by abundant laboratory findings in which constriction of either or both ventricles is held to be of primary importance, whereas constriction of the auricles was rarely found to be significant. In making application of their concept in the surgical correction of constrictive pericarditis, they suggest that total excision of the pericardial

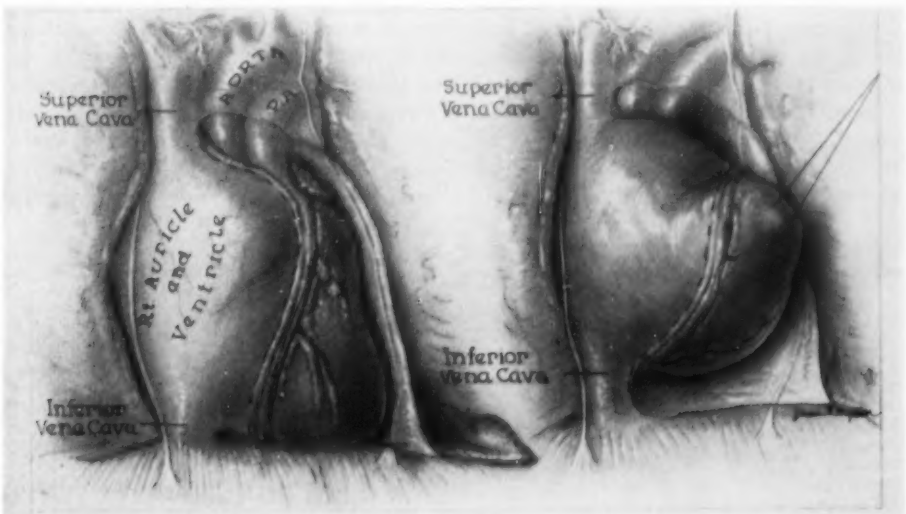
From the Laboratory for Surgical Research, Stanford University School of Medicine. This work was supported by a grant from the Life Insurance Medical Research Fund.

Boyd Foundation (Long Beach, Calif.) Fellow in Thoracic Surgery (Dr. Parsons).

* References 4, 10, 11, 13, 16, 18, 20, 22, 26, 27, 28, 29.

† References 1, 2, 3, 7, 12, 15, 19, 23, 30, 32.

Fig. 1.—The pericardium over the left heart was excised and the edge of the remaining pericardium sutured securely to form an enclosed sac over the right heart.



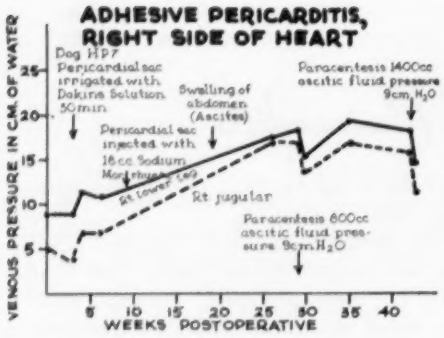

ADHESIVE PERICARDITIS, RIGHT SIDE OF HEART
Dog HP7
Pericardial sac irrigated with Dakins Solution 30 min
Swelling of abdomen (Ascites)
Paracentesis 1400cc ascitic fluid pressure 9cm. H2O
Pericardial sac injected with 18 cc Sodium Mordhuate 1/40
Rt lower leg
Rt jugular
Paracentesis 800cc ascitic fluid pressure 9cm.H2O
VENOUS PRESSURE IN CM. OF WATER
WEEKS POSTOPERATIVE

Fig. 2 (Animal HP 7).—Marked rise in venous pressure in both the jugular vein and the veins of the lower leg occurred after constrictive pericarditis limited to the right heart.

scar is to be desired, but that liberation of both ventricles is essential.

The following experimental study was made for the purpose of further clarifying the surgical approach to the treatment of chronic constrictive pericarditis.

CONSTRICTION OF RIGHT SIDE OF HEART

With use of mongrel dogs, intravenous pentobarbital sodium (Nembutal) anesthesia, and endotracheal positive pressure respiration, the heart was exposed through a median sternotomy. The pericardium over the left side of the heart was excised. The edge of the retained pericardium was sutured with a continuous fine nonabsorbable surgical (silk) suture (Fig. 1) to the scarified surface of the superior vena cava, the aorta, the pulmonary conus, along the border of the right ventricle immediately to the right of anterior descending coronary artery, the posterior surface of the heart, and the inferior vena cava. The pericardium thus

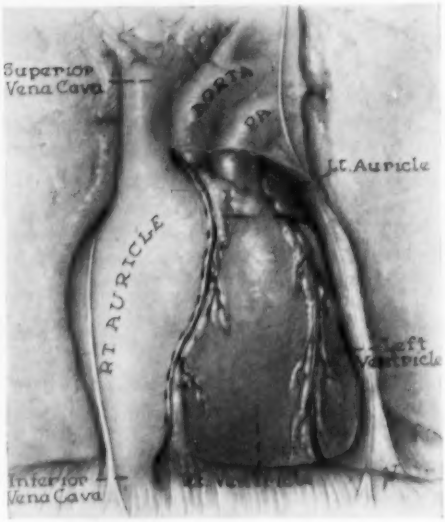


Fig. 3.—The pericardium over the left heart and right ventricle was excised and the remaining pericardium sutured to form an enclosed sac over the right auricle and auricular appendage.

formed an enclosed sac over the structures of the right heart, leaving the left heart free of pericardium.

Four weeks were allowed to elapse, permitting the pericardium to heal and to form a water-tight sac over the right heart (Animal HP 7). The heart was again exposed through a right intercostal incision and the pericardial sac irrigated with surgical solution of chlorinated soda (Dakin's solution) for 30 minutes. One month later, the sac was injected with 18 cc. of sodium morrhuate. Immediately after the introduction of irritants into the pericardial sac, there was a rise in the venous pressure above 15 cm. of water in both the jugular vein and the veins

Findings on Cardiac Catheterization Studies

Segmental Constriction	Weight, Kg.	Pressures: Cm. H_2O Jugular Vein	Cm. H_2O Femoral Vein	Mm. Hg Right Ventricle	Mm. Hg Pulmonary Artery	Mm. Hg Left Ventricle	Mm. Hg Aorta	Cardiac Output, Cc/Min. 20.9% O_2	Cardiac Output, Cc/Min. 14.8% O_2
Right side, heart.....	17	14	15	30/6	10/3	80/0	85/55	582	502
Right auricle.....	18	4	8	25/0	25/10	140/0	140/90	1200	1402
Inferior vena cava....	12	8	22	42/0	30/10	108/0	145/98	...	...
Left	11.8	5	8	35/0	20/5	....	160/120	1549	298
Side	22.4	7	9	35/0	20/10	140/0	138/120	2982	1919
Heart	14	2	4	20/0	15/5	160/0	140/110	401	1351
Left auricle.....	11.5	10	7	....	....	180/0	178/140	...	...

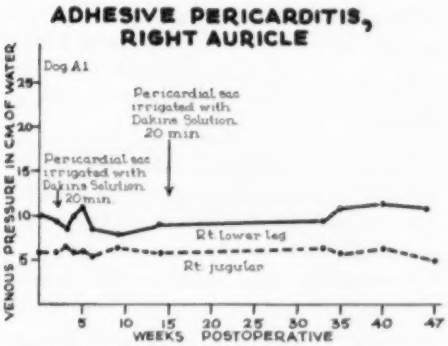

ADHESIVE PERICARDITIS, RIGHT AURICLE
Dog A1
Pericardial sac irrigated with Dakins Solution. 20 min.
Pericardial sac irrigated with Dakins Solution. 20 min.
Rt lower leg
Rt jugular
VENOUS PRESSURE IN CM OF WATER
WEEKS POSTOPERATIVE

Fig. 4 (Animal HP 6).—Mild rise in the venous pressure of both the jugular vein and the veins of the lower leg occurred after constrictive pericarditis limited to the right auricle.

of the lower leg (Fig. 2). By the 20th postoperative week, 1400 cc. of ascitic fluid was removed, and the animal was killed. The right ventricle and auricle were small and constricted, while the left ventricle was large and nonconstricted. Pleural effusion was absent.

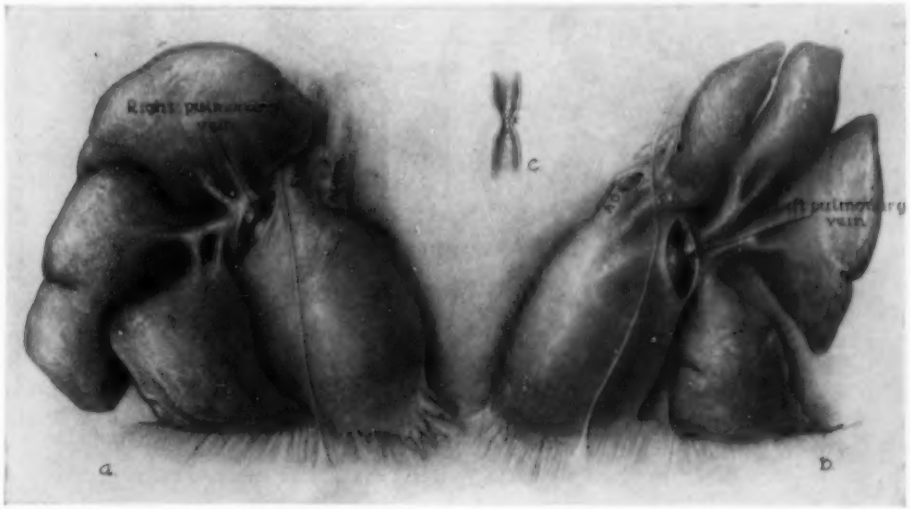
In a second animal (HP 25), weighing 17.0 kg. and treated identically as the animal above, ascites failed to develop, although there was a marked rise in the pressure in the jugular vein to 14 cm. of water and in the lower leg veins to 15 cm. of water. Two years later the venous pressures remained the same. Cardiac catheterization was performed and the pressures recorded (Table 1). The right ventricule (30/6 mm. Hg) showed a slight rise in the diastolic pressure above normal. The pulmonary artery (10/3 mm. Hg), the aorta (80/55 mm. Hg), and the left ventricle (80/0 mm. Hg) all showed a drop in systolic pressure. There was a narrowing of the aortic pulse pressure. Cardiac output, by the direct Fick[14] method, was low (582 cc/min.).

Results.—Constriction of the right side of the heart produces elevated peripheral venous pressure, ascites, elevated right ventricular diastolic pressure, lowered aortic pressure and pulse pressure, and a diminished cardiac output.

CONSTRICTION OF RIGHT AURICLE

With use of the same technique as above, the pericardium was cut and sutured so as to form an enclosed pericardial sac limited to the right auricle and auricular appendage (Fig. 3). The pericardium was cut away completely from the remaining heart. Again, four weeks was allowed to elapse for the pericardial sac to heal and to become watertight before injection of irritants. Over a

Fig. 5.—The main branch of the right pulmonary vein (*a*) and the left pulmonary vein (*b*) were isolated intrapericardially and constricted to varying degrees with an umbilical tape ligature (*c*).



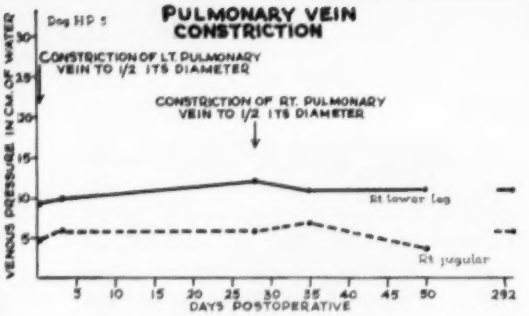

Dog HP 5
PULMONARY VEIN CONSTRICTION
CONSTRICTION OF LT. PULMONARY VEIN TO 1/2 ITS DIAMETER
CONSTRICTION OF RT. PULMONARY VEIN TO 1/2 ITS DIAMETER
VENOUS PRESSURE IN CM. OF WATER
Rt lower leg
Rt jugular
DAYS POSTOPERATIVE

Fig. 6 (Dog HP 5).—No rise in the venous pressure occurred in either the jugular vein or the veins of the lower leg after constriction of the main pulmonary veins in two stages, 30 days apart.

period of 40 weeks there was a very mild rise in the venous pressure of both the jugular vein and the veins of the lower leg (Fig. 4). At the time of autopsy, there was no evidence of ascites or pleural effusion. The auricular appendage was completely obliterated and the small right auricle was found to be covered by a thick constricting pericardium. On cut surface, the wall of the auricle was thickened but the lumen appeared large enough to conduct the blood adequately.

Constriction of the right auricle was similarly produced in another animal (HP 17), weighing 18 kg. The venous pressure in the jugular vein (4 cm. H_2O) and veins of the lower leg (8 cm. H_2O) remained unelevated after two and one-half years. Cardiac catheterization showed no abnormal pressures (right ventricle 25/0 mm. Hg; pulmonary artery 25/10 mm. Hg; aorta 140/90 mm. Hg, and left ventricle 140/0 mm. Hg). Cardiac output was normal (1200 cc/min.) (Table).

Results.—Constriction of the right auricle produced no signs or symptoms of constrictive pericarditis. The lumen of the auricle has remained adequate to conduct the flow of blood to the right ventricle. The anterior and lateral portions of the right auricle are covered by pericardium. The portions bounded by the interauricular septum, the transverse sinus, the right ventricle, and the tricuspid valve are not covered with pericardium and are therefore not subjected to constriction by this structure. Since only one-half or less of the right auricle is covered by pericardium, it seems unlikely that its lumen could be narrowed by the constricted pericardium sufficiently to produce symptoms. Obstruction to the flow of blood into the right ventricle probably occurs when the fibrosis of the pericardium results in a constricting band around the auriculoventricular groove. We were unable to demonstrate this process experimentally.

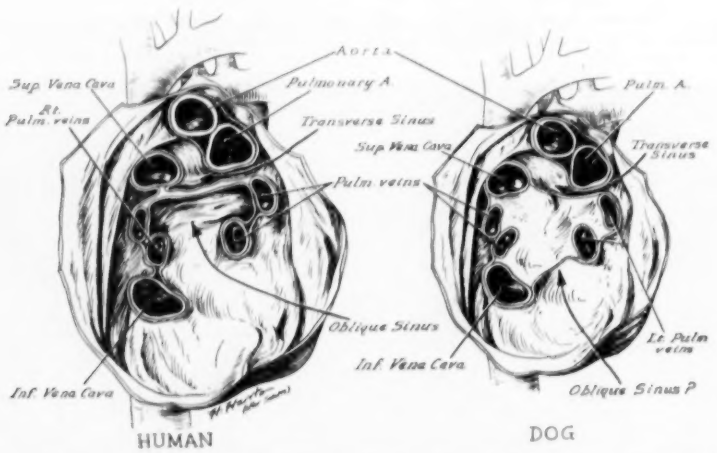


Fig. 7.—The heart has been cut away leaving the posterior pericardium and its reflection around the great vessels. In the human, owing to the presence of the oblique sinus, the pericardium completely surrounds the inferior vena cava, superior vena cava, and the pulmonary veins. The oblique sinus is not developed in the dog, thereby limiting the pericardial covering of the circumferences to two-thirds around the inferior vena cava and superior vena cava and to less than one-half around the pulmonary veins.

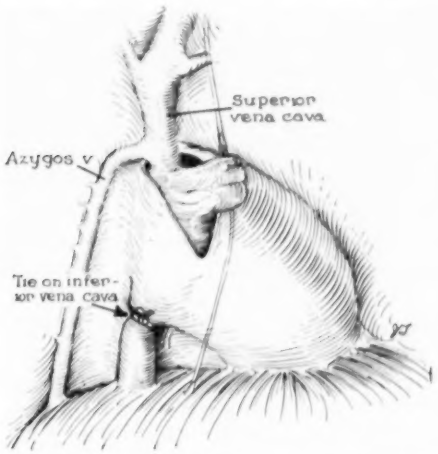


Fig. 8.—The inferior vena cava was constricted by an umbilical tape ligature to one-half its outside diameter at the pericardial reflection.

CONSTRICTION OF THE PULMONARY VEINS

The pulmonary veins were constricted in five animals. In four, the heart was exposed through a median sternotomy. The main branch of both pulmonary veins was exposed and freed intrapericardially by incising the pericardium parallel and posterior to the phrenic nerves (Fig. 5). In three animals the pulmonary veins were constricted to one-half their outside diameter bilaterally by an umbilical tape ligature. One animal died immediately after surgery, and the other two died on the second postoperative day. No anatomical cause of death was found in one animal. The other animal had marked pulmonary edema and dyspnea and coughed up bloody, frothy sputum. Fifteen cubic centimeters of sanguinous fluid was present in both sides of the chest. In the fourth animal (HP 2), both pulmonary veins were constricted to two-thirds of the outside diameter, i. e., the diameter of the veins was reduced by one-third. This animal was followed 85 days, and there was no rise in the peripheral venous pressure of the jugular or saphenous veins. At autopsy there was no pleural effusion or ascites.

In the fifth animal (HP 5), the pulmonary veins were exposed through right and left intercostal incisions in two stages. The left pulmonary vein was constricted to one-half its diameter, and 30 days later the right pulmonary vein was constricted to one-half its diameter. There was no rise in the peripheral venous pressure in the jugular or saphenous veins (Fig. 6). The animal was killed in 292 days, and there was no pleural effusion or ascites.

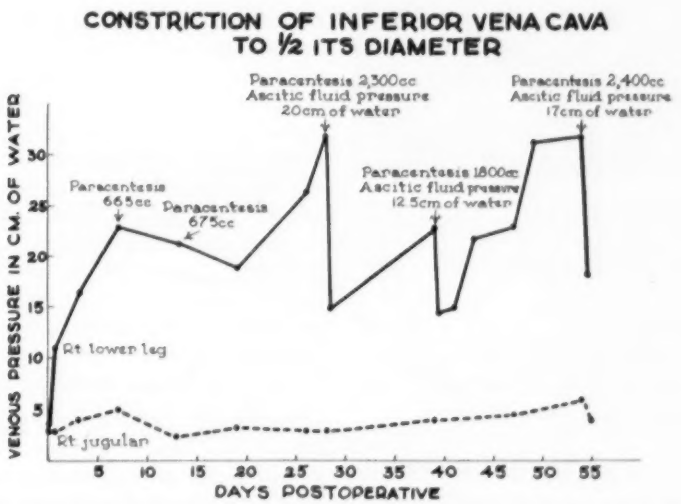


Fig. 9 (Animal VC 1).—After constriction of the inferior vena cava to one-half its diameter, there was an immediate rise of the venous pressure of the lower leg, while that of the jugular vein remained unchanged. By the seventh postoperative day, permanent ascites was present.

Results.—Acute constriction of the pulmonary veins resulted in pulmonary edema, hydrothorax, and death. However, chronic constriction of the pulmonary veins failed to show pulmonary edema, hydrothorax, or a rise in the peripheral venous pressure. Less than one-half of the diameter of the pulmonary veins of the dog is enclosed in the pericardial sac (Fig. 7). It is unlikely they would be constricted significantly by peri-

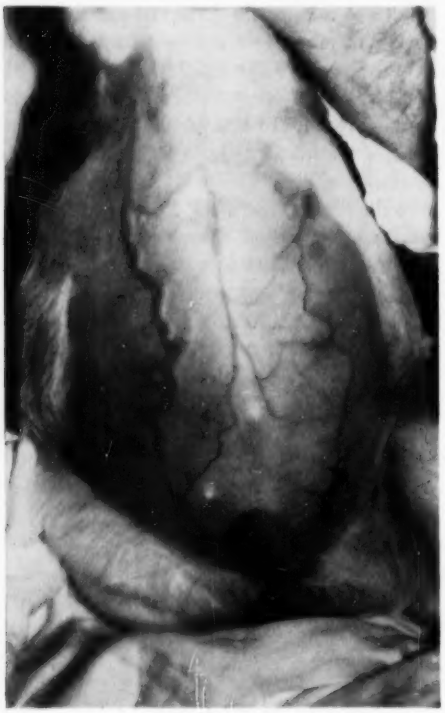


Fig. 10 (Animal VC 1).—An infra-red photograph demonstrates the dilated and tortuous superficial veins of the abdominal wall and the distended abdomen, 26 days after constriction of the inferior vena cava.

cardial scarring. In the human (Fig. 7), however, owing to the presence of the oblique sinus, the pulmonary veins are almost completely surrounded by pericardium.

CONSTRICTION OF THE VENAE CAVAE

The inferior vena cava was exposed through a right intercostal incision and was constricted with an umbilical tape ligature at the pericardial reflection to one-half its outside diameter (Fig. 8). In the first animal (VC 1), there was an immediate rise in the venous pressure of the lower leg, while the pressure in the jugular vein remained normal (Fig. 9). Appetite was poor, and markedly dilated superficial veins appeared on the abdominal wall. By the seventh postoperative day, the venous pressure of the lower leg had risen from 3 to 23 cm. of water, and definite signs of ascites were present (Fig. 10). Twenty-three hundred cubic centimeters of straw-colored fluid was removed by paracentesis. The animal was killed and the venous system visualized by roentgenogram after injection of the femoral vein with bismuth oxychloride.[21] An extensive collateral venous bed was demonstrated (Fig. 11).

In a second animal (VC 5), constriction of the inferior vena cava to one-half its diameter was again produced. Again, the venous pressure of the right lower leg rose above 15 cm. of water, and the animal developed ascites. Following a peak venous pressure of 20 cm. of water on the 12th postoperative day, there was a gradual decline in the pressure, reaching a slightly elevated level on about the 40th postoperative day (Fig. 12). Ascites behaved similarly. On the 20th postoperative day, the ascites had begun to decline; by the 100th day the physical signs of ascites had disappeared, and by the 140th day ascites fluid could not be obtained by a diagnostic paracentesis (Fig. 13). Through a second thoracotomy, on the 140th day, the superior vena cava was constricted between the azygos vein and the right auricle to two-thirds its diameter (Fig. 14). An immediate rise of venous pressure in the jugular vein from 5 to 32 cm. of water and in the lower leg from 16 to 26 cm. of water occurred (Fig. 12). The ascites reappeared by the fifth postoperative day (Fig. 13).

Ascites was similarly produced in a third animal (HP 16). Venous pressures (Table) were normal in the jugular vein (8 cm. H_2O), and markedly elevated in the veins of the lower leg (22 cm. H_2O). Normal intracardiac pressures were found upon

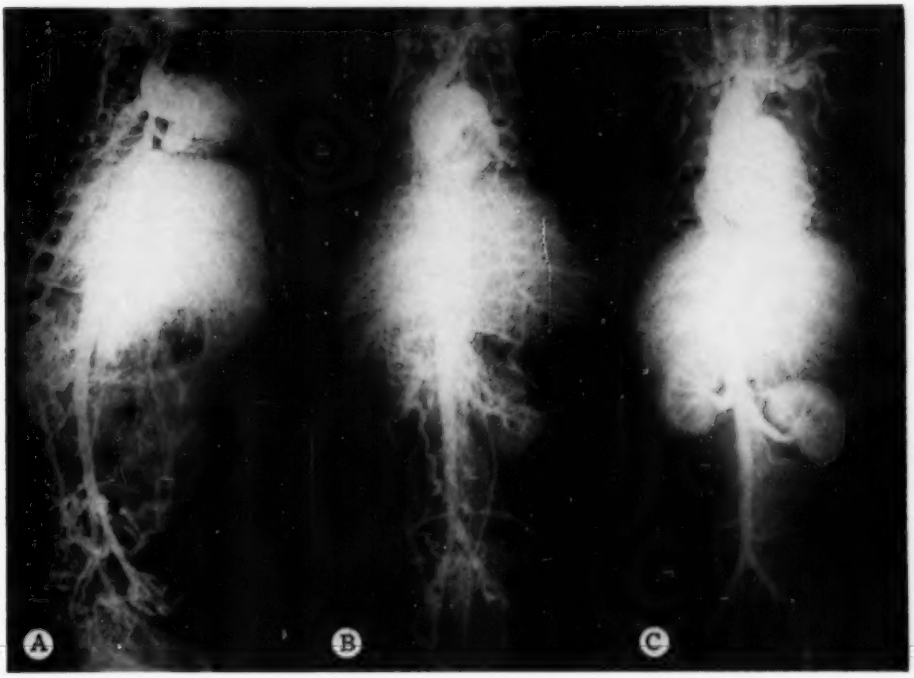


Fig. 11.—(*a*) and (*b*) (Animal VC I). X-rays of the injected venous system of an animal two months after constriction of the inferior vena cava to one-half its diameter demonstrates the markedly dilated collateral veins; (*c*) (Animal ST I), and x-ray demonstrates the injected venous system of a normal animal.

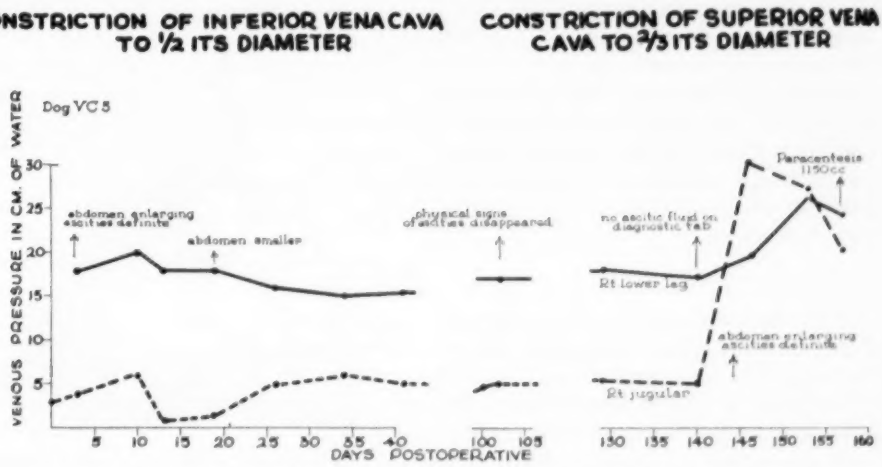

CONSTRICTION OF INFERIOR VENA CAVA TO 1/2 ITS DIAMETER
CONSTRICTION OF SUPERIOR VENA CAVA TO 2/3 ITS DIAMETER
Dog VC 5
VENOUS PRESSURE IN CM. OF WATER
30
25
20
15
10
5
abdomen enlarging ascites definite
abdomen smaller
physical signs of ascites disappeared
no ascitic fluid on diagnostic tap
Paracentesis 1150cc
Rt lower leg
abdomen enlarging ascites definite
Rt jugular
5 10 15 20 25 30 35 40 100 105 130 135 140 145 150 155 160
DAYS POSTOPERATIVE

Fig. 12 (Animal VC 5).—After constriction of the inferior vena cava to one-half its diameter, there was an immediate rise of the venous pressure of the lower leg and the development of ascites, followed by a gradual fall in venous pressure and the disappearance of the ascites. After partial constriction of the superior vena cava the venous pressure of the lower leg again rose and the ascites reappeared.

Fig. 13.—(*a*) (Animal VC 5) One hundred forty days after constriction of the inferior vena cava, all evidence of ascites had disappeared; (*b*) on the 18th day after constriction of the superior vena cava, ascites reappeared.

cardiac catheterization of the right ventricle (42/0 mm. Hg), pulmonary artery (30/0 mm. Hg), left ventricle (108/0 mm. Hg), and aorta (145/98 mm. Hg).

Results.—Constriction of the inferior vena cava by one-half its diameter resulted in a rise in peripheral venous pressure, dilated superficial veins, ascites, an extensive venous collateral circulation, and normal intracardiac pressures.

CONSTRICTION OF LEFT SIDE OF HEART

With the same technique as described above for constriction of the right side of the heart, three animals were prepared with the constricting pericardium limited to the left side of the heart, including the left ventricle and auricle (Fig. 15). The animals lived normal, healthy lives for three years. Ascites, pleural effusion, pulmonary edema, and peripheral venous congestion did not occur. Elevation of the venous pressures in the jugular vein or the veins of the lower leg did not occur. Cardiac catheterization studies were done (Table). The right ventricular, pulmonary arterial, and left ventricular pressures were normal. The aortic systolic pressure was not lowered; however, the pulse pressures were narrowed. Two of the animals had normal cardiac outputs. The third animal (HP 15) showed a lowered cardiac output while breathing normal air (20.9% oxygen). However, the animal was able to raise the output to normal when breathing air with a diminished oxygen content (14.8% oxygen).

Lyons and Burwell[24] have demonstrated in constrictive pericarditis the increase in the cardiac output with an increase in the heart rate. The stroke volume may be diminished, but within limits tachycardia will increase the cardiac output.

At autopsy, the hearts showed constriction of the left ventricles and auricles by a thick, constricting pericardium. Ascites was not seen. Bilateral pleural effusion of approximately 80 to 100 cc. was found in HP 15. There were no gross changes in the lungs or liver.

Results.—Constriction of the left side of the heart resulted in minimal pleural effusion,

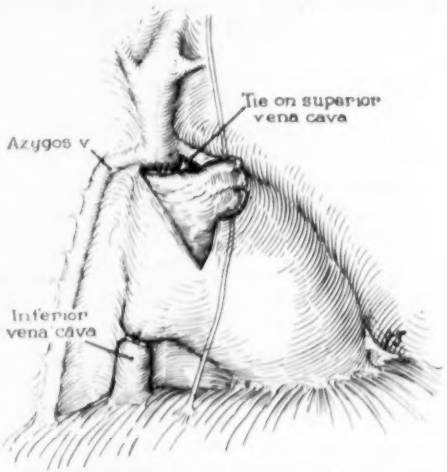


Fig. 14 (Animal VC 5).—One hundred forty days after constriction of the inferior vena cava, the superior vena cava was constricted between the azygos vein and the right auricle by one-third, leaving two-thirds of its diameter patent.

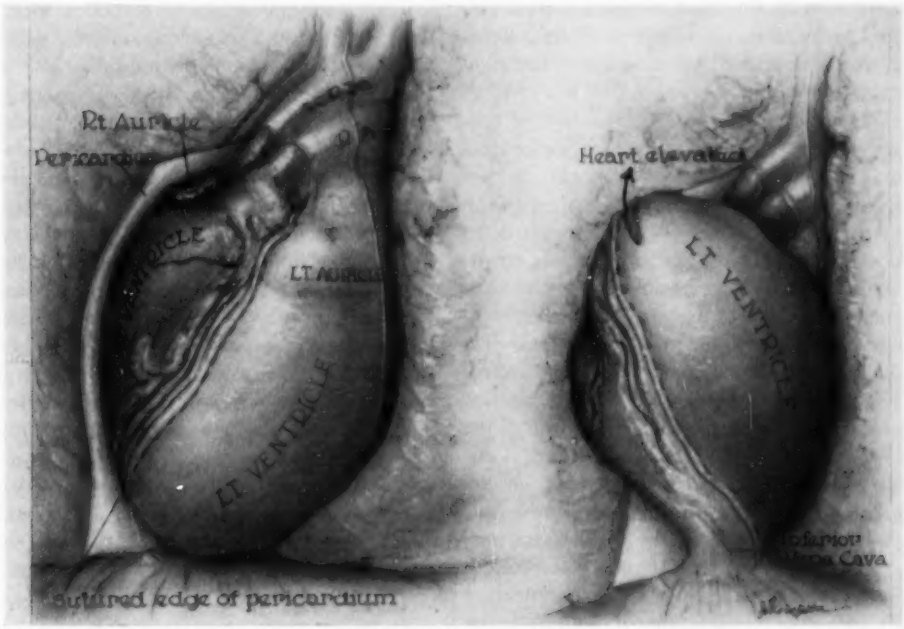


Fig. 15.—The pericardium over the right heart was excised and the edge of the remaining pericardium sutured securely, to form an enclosed sac over the left heart.

diminished aortic pressure, and a decrease in cardiac output.

CONSTRICTION OF THE LEFT AURICLE

The left auricle was constricted, with the use of the same technique as described above. Ascites, pleural effusion, pulmonary edema, and peripheral venous congestion were not seen. The animal lived a normal and healthy year until the termination of the experiment. Venous pressures (Table) were not elevated in the jugular vein (10 cm. H_2O) or in the veins of the lower leg (7 cm. H_2O). An elevation of the left ventricular (180/0 mm. Hg) and aortic (175/140 mm. Hg) pressures was found on cardiac catheterization. Catheterization of the pulmonary artery was not accomplished. At autopsy, the constriction of the auricle appeared similar to the right auricle.

Results.—Constriction of the left auricle produced no signs or symptoms of constrictive pericarditis. The anterior and lateral portions of the left auricle are covered with pericardium. The portions of the left auricle bounded by the transverse sinus, the interauricular septum, the left ventricle, and the mitral valve are not covered with pericardium. In the dog the oblique sinus is very small or nonexistent, so that no free pericardium is present in this area as in the human with a well developed oblique sinus (Fig. 7). In both the human and the dog, the pericardium does not cover more than one-half of the surface of the left auricle, and it therefore seems unlikely that constriction of the pericardium would narrow the lumen sufficiently to produce symptoms.

COMMENT

Constriction of the right side of the heart produces the majority of the signs and symptoms usually associated with the diagnosis of constrictive pericarditis. An increase in the venous pressure with congestion of the jugular veins and veins of the lower leg, ascites, edema, and a diminished cardiac output are usually seen. Experimental constriction of the right auricle produced none of these signs or symptoms. Pericardectomy

must liberate the right ventricle if constricting pericardium is found over this structure. Experimental constriction of the right auricle and pulmonary veins did not produce signs of decompensation. However, a constricting ring, if found in the nearly encircling auriculoventricular groove should be removed or divided so as to allow expansion of the orifice of the valve and unobstructed flow of blood from the right auricle to the ventricle. We were unable to demonstrate such a constriction in our experiments.

Constriction of the venae cavae will produce symptoms of constrictive pericarditis. Experimental constriction of the inferior vena cava produces venous congestion of the lower extremities, edema and ascites.‡ Constriction of the superior vena cava on the cardiac side of the azygos vein produces pleural effusion and edema and venous congestion of the upper extremities and head.§ Bolton,[6] in 1917, found in experimenting on cats that constriction of both superior and inferior vena cava led to a condition of dropsy similar in its distribution to that produced by pericardial constriction. At operation, we have found the inferior vena cava markedly constricted by scar in direct continuation with the fibrotic and calcified pericardium which securely anchored the heart along its inferior border to the diaphragm.[22] The following is a quotation in which Beck and Griswold[2] describe the necessity of freeing the scar around the inferior vena cava in one of their experimental animals.

> The exposure through the left side of the chest was inadequate to permit complete resection of the pericardium. The region where the venae cavae entered the right auricle was inaccessible through this exposure. A striking improvement followed the operation. During the succeeding three weeks, however, decompensation developed. An operation was again carried out. An exposure was made through the right side of the chest. A segment of thickened pericardium was found adherent to the heart where the venae cavae entered the right auricle, and a band of thickened pericardium caused a partial obstruction to the inferior vena cava. This portion of the pericardium was excised and the circulation again improved.

Pericardectomy must be extended to include the pericardium surrounding the venae cavae if it is found constricting these vessels. If only the inferior vena cava is partially obstructed, the development of collateral venous circulation will direct the blood through a dilated superior vena cava, thereby reducing the pressure in veins of the legs and lower trunk and causing the ascites to disappear (Animal VC 5). Perhaps the delay in reducing the venous pressure and the disappearance of ascites in some of the operated cases is contingent on the time necessary for the development of collateral circulation following limited pericardectomy. The inferior vena cava is more frequently constricted than the superior vena cava, probably as a result of the upright position which allows the toxic material during the period of pericardial effusion to collect around this structure.

Constriction of the left side of the heart is different from that of the right side. Peripheral venous congestion, edema, and ascites do not occur. Pulmonary hypertension, rales, dyspnea, and an accentuated second pulmonic sound may develop.‖ Diminished cardiac output and a narrowed pulse pressure were seen. Constriction of the left auricle produces no measurable difficulty.

Decortication must include both ventricles, the venae cavae, and the auriculoventricular grooves if these structures are found to be invested by the constricting pericardium.

SUMMARY

Venous pressures, cardiac catheterizations, and cardiac output studies were made on dogs with constriction of anatomic segments of the heart.

Constriction of the pericardium limited to the right side of the heart produced a marked rise in the peripheral venous pressure, ascites, and a diminished cardiac output.

Constriction of the pericardium limited to the right auricle produced no rise in the peripheral venous pressure, no ascites, no

‡ References 6 and 33.

§ References 9, 5, and 17.

‖ References 8, 23, 25, and 31.

alteration in the cardiac catheterization pressures, and no change in the cardiac output.

Constriction of the pulmonary veins by varying degrees up to the point of death has not produced a rise in peripheral venous pressure and ascites or pleural effusion.

Obstruction of the inferior vena cava by one-half produced markedly dilated superficial veins, extensive collateral venous circulation, elevated venous pressure, and marked ascites in seven days. If the animal was permitted to survive, the ascites gradually disappeared, only to reappear when the superior vena cava was constricted by one-third its outside diameter. This demonstrates the great importance of collateral circulation by way of the superior vena cava in preventing or correcting ascites when the inferior vena cava is obstructed.

Constriction of the left side of the heart did not produce peripheral venous congestion, edema, or ascites. Diminished cardiac output and narrowed pulse pressure were observed.

Constriction of the pericardium limited to the left auricle produced no rise in the peripheral venous pressure, no ascites and no alteration in the cardiac catheterization pressures.

Decortication must include both ventricles, the venae cavae, and the auriculoventricular grooves if these structures are found to be invested by the constricting pericardium.

Mr. Dusan Surman, laboratory technician, gave technical aid in the preparation of his paper.

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Observations on the Growth of Aortic Anastomoses in Puppies

III. Use of Absorbable Gelatin Sponge (Gelfoam) in Relation to Growth

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Absorbable gelatin sponge (Gelfoam) has gained wide acceptance as an "absorbable hemostatic packing" in a large number of situations involving vascular suture lines and the control of bleeding in various parts of the body. Evidence has been presented previously indicating that the absorbability of this substance may vary depending on the individual circumstances attending its use. The organization of absorbable gelatin sponge wrapped around fabricated autogenous arterial grafts placed in the aorta results in the formation of a dense fibrous sheath surrounding and supporting the transplant.[1] Such a development, while beneficial in many situations, would constitute a distinct disadvantage in terms of a vascular anastomosis in a young subject, where an increasing diameter at the suture line commensurate with the growth of the individual might be of real importance. The present study was designed to determine whether or not absorbable gelatin sponge interferes with the growth of aortic anastomoses in puppies.

EXPERIMENTAL METHOD

This has been described in detail in previous publications.* Mongrel puppies were utilized, ranging in weight from 6 to 11.5 lb., with an average weight of 8 lb. General anesthesia was obtained by the intraperitoneal administration of small amounts of a standard veterinarian solution of pentobarbital (Nembutal), supplemented as needed by ether inhalation. The peritoneal cavity was entered through a vertical midline incision. The abdominal aorta was mobilized for a short distance above the trifurcation and transected between seraphine clamps. The diameter of the pulsating aorta varied from 4 mm. in the smaller puppies to one of 5.5 mm. in a 10-lb. pup. The ends of the divided vessel were irrigated with saline solution and united with simple interrupted 00000 medium chromic absorbable (gut) sutures.† The anastomosis usually required eight sutures, consisting of one at each lateral angle and three each for the anterior and posterior walls. There was no attempt at eversion, and all knots were placed on the outside of the vessel. On release of the occluding clamps the immediate ooze was controlled by gentle pressure over a strip of compressed absorbable gelatin sponge, size 100,‡ which had been placed around the anastomosis. The abdominal wall was closed in layers, and penicillin, 300,000 units, was given intramuscularly daily for the first three days.

The use of suture material armed at each end with an Atraumatic fine needle has greatly facilitated surgical repair of vessels of this small caliber. By passing the needle from inside out at each end of the collapsed vessel, it is possible to include the full thickness of the vessel wall with each bite and avoid the hazard of including a few strands of the opposite wall.

Dogs were maintained for survival studies extending for as long as 467 days, including the period of maximum and most rapid growth. Retrograde aortography was performed early in the postoperative course and prior to killing, by the technique previously described.[3] Direct measurements of the aorta were made in the living animal whenever possible at the end of the growth period, and an autopsy was performed in every instance. In the

From the Surgical Research Laboratory, the Montefiore Hospital.

Aided by a grant from the Playtex Park Research Institute.

* References 2 and 3.

† Supplied by the Ethicon Suture Laboratories, Inc., New Brunswick, N. J.

‡ Supplied by the Upjohn Company, Kalamazoo, Mich.

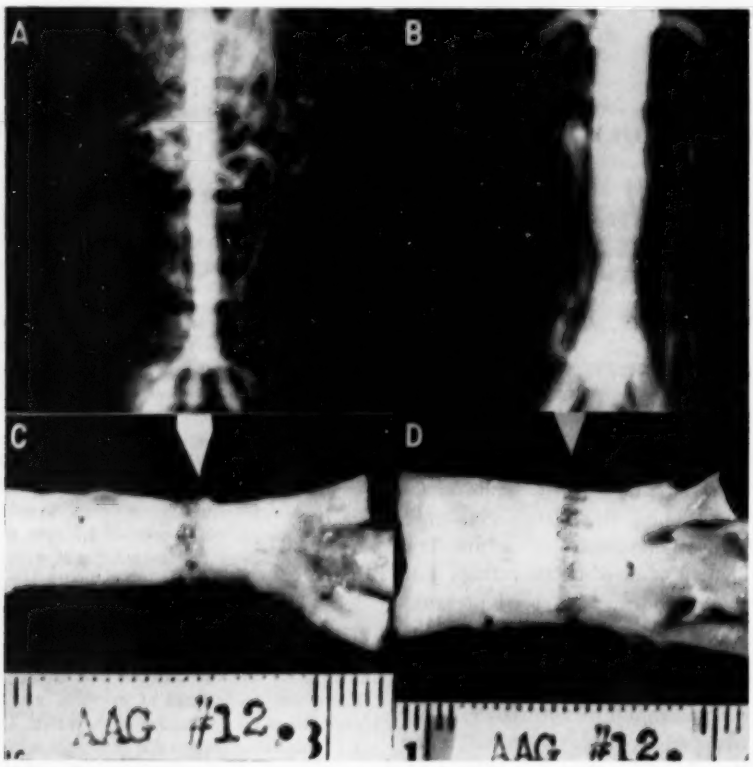


Fig. 1.—Dog A. A. G. 12. Aortograms 31 (*A*) and 288 (*B*) days postoperatively, and gross appears of specimen (*C, D*). Narrowing apparent only in *B*.

gross examination, the comparative diameter of the anastomosis and the aorta, the degree of periaortic reaction, and any evidence of residual gelatin sponge were carefully determined. Sections of the formalin-fixed material, stained with hematoxylin and eosin, were obtained.

EXPERIMENTAL RESULTS

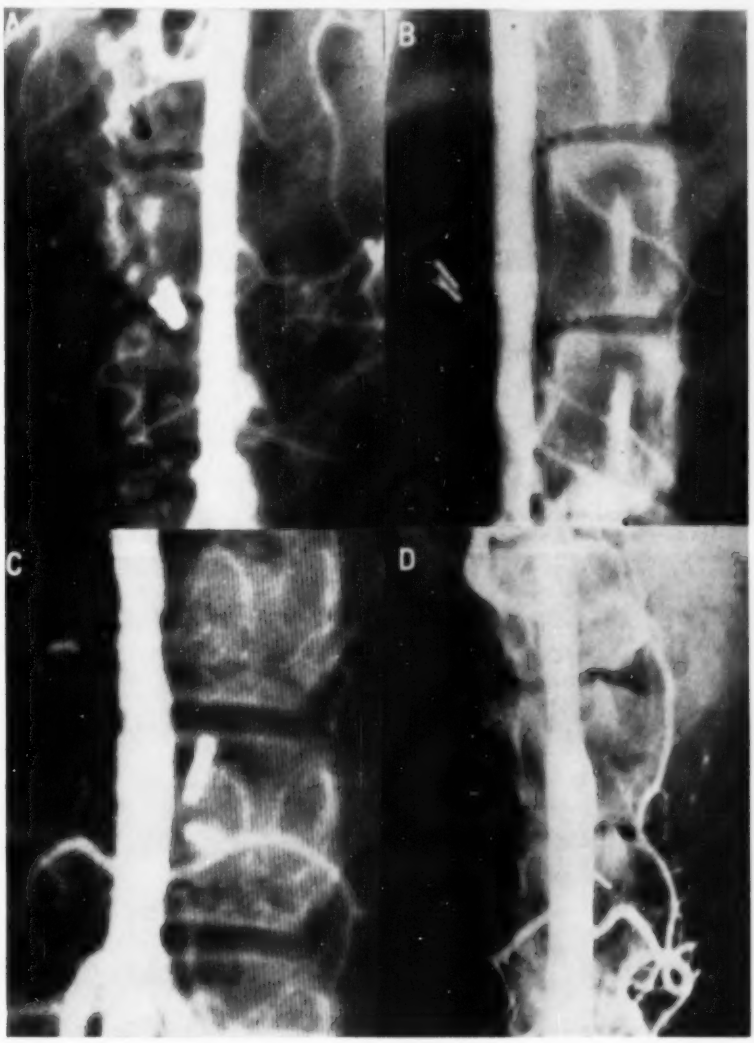
The present group consists of 14 puppies. Of these, four survived to adult size with patent anastomoses. Loss of the other 10 animals, owing to distemper, anesthesia, and occluded vessels, was comparable to that in the control series of 27 puppies in which absorbable gelatin sponge was not employed,[3] and cannot be attributed to the use of this substance. The difficulties in maintaining puppies under the conditions of an unstable experimental colony account for the small size of the series. The status of the gelatin sponge-wrapped aortic wall in animals dying or killed within a few months of operation was also of interest in comparison with the end-results.

The pertinent data for the survival animals are summarized in the Table. In no instance was the diameter of the anastomosis less at the time of killing than at the time of operation. In all of the animals surviving through the growth period there was an appreciable increase in the diameter at the anastomosis, although this was slightly less than that for the remainder of the aorta. There was no greater degree of periaortic reaction than in the control animals, and no Gelfoam was grossly identifiable as such. Brown-stained aggregates of lymphoid tissue were observed near the terminal end of the aorta in some autopsies. These were interpreted grossly as lymph nodes containing assimilated gelatin sponge and were removed for further study.

Dog No.	Weight, Lb.		Survival Period (Days)	Diameter of Aorta* (Mm.)		Specimen Opened
	Initial	Final		Operation	Killing	
1	8	38	467	(1) 5 (2) 5	7 9	Slight constriction; postanastomotic dilatation; sutures not identifiable
2	11.5	54	407	(1) 5 (2) 5.5	6 8	Slight constriction; postanastomotic dilatation; sutures identifiable
3	10	55	384	(1) 5 (2) 5.5	6 8	Slight constriction; postanastomotic dilatation; sutures identifiable
6	8	10	72	(1) 5 (2) 5	5 5	No narrowing (distemper)
12	7.5	30	323	(1) 4 (2) 4	6.5 7	No narrowing; very low anastomosis; sutures identifiable
13	7	9	61	(1) 4 (2) 4	4 4	No narrowing (distemper)

* Refers to diameter (1) at anastomosis; (2) above and below anastomosis.

Fig. 2.—Comparative aortograms 10 (*A*) and 315 (*B*) days postoperatively in control (AA 12), and 6 (*C*) and 396 (*D*) days postoperatively in absorbable gelatin sponge series (AAG 2). Postanastomotic dilatation in latter.



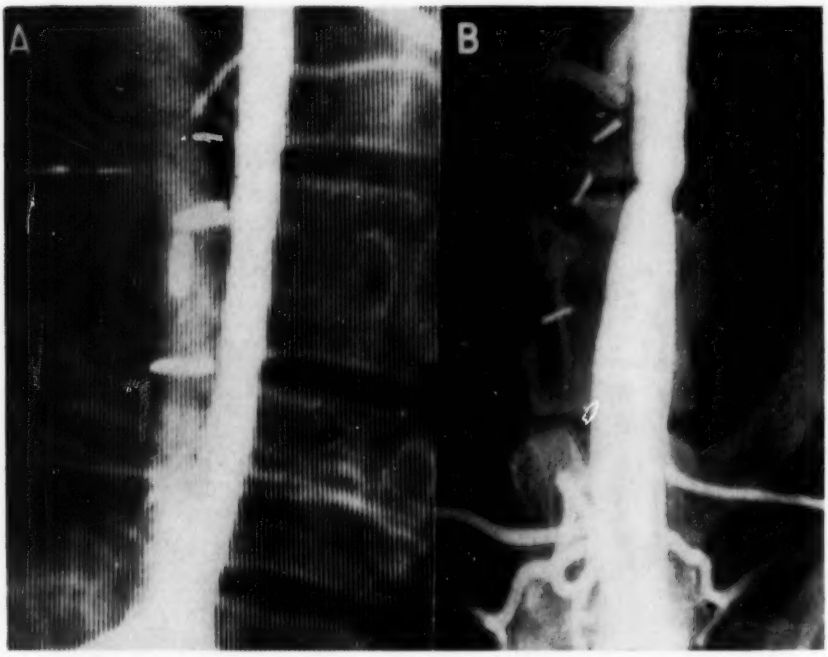


Fig. 3.—Dog A. A. G. 3. Aortograms 7 (*A*) and 315 (*B*) days postoperatively, with postanastomotic dilatation in *B*.

The superiority of aortography over gross measurements in evaluating the functional status of a vascular suture line is clearly demonstrated by the distinct narrowing in the late aortogram in dog No. 12 (Fig. 1*B*), in contrast to the uniform caliber in the early films and the postmortem specimen (Fig. 1*A*, *C*, *D*). It is only by having available aortograms taken early postoperatively, for comparison with the films in the adult ani-

Fig. 4.—Control specimen (AA 17) 194 days and absorbable gelatin sponge specimen (AAG 3) 384 days postoperatively, showing postanastomotic dilatation in latter.

mal (as in Figs. 1, 2, and 3), that one can define whether a late constriction is due to a lag in growth or to a persistence of a defect created at the time of surgery. The puppy aortograms in these figures have been enlarged to focus on the anastomoses.

Slight degrees of postanastomotic dilatation were present in every instance, with the exception of the very low anastomosis referred to in the preceding paragraph. This is in striking contrast to the uniform contours of the aortas in the control series. A comparison of the early and late aortograms in control and gelatin sponge-wrapped anastomoses is demonstrated in Figure 2. The contrast is evident again in Figures 3 and 4, in which the aortographic and gross appearance of comparable specimens confirms the dilatation that develops distal to even this minimal amount of constriction in the growing animal.

Microscopically, the gelatin sponge-wrapped anastomoses were not significantly thicker than the controls. Residual absorbable (gut) suture was seen in the specimens in both series, with a foreign body reaction that was minimal and comparable in both groups. A slight increase in the amount of fibrous tissue in the adventitia at the anastomoses was present in the gelatin sponge series. Identifiable fragments of absorbable gelatin sponge were found in specimens at 8, 61, and 72 days after operation. No particles of absorbable gelatin sponge could be demonstrated in the hyperplastic periaortic lymph nodes, in which large macrophages were prominent.

COMMENT

The comparatively small increments of growth demonstrable in the aorta of the dog impose certain limitations on a study of this nature. A four- or five-fold increase in the size of the animal may be accompanied by a difference of only a few millimeters in aortic diameter. In a previous study, comparing the relative effects of interrupted nonabsorbable (silk) and absorbable (gut) sutures on the growth rate of vascular anastomoses in puppies, it was determined that growth was satisfactory and approximately the same with either method.[3] The only difference between the previous absorbable (gut) group and the present series is the addition of absorbable gelatin sponge around the suture line.

It would appear from a study of the Table that the growth rate of the gelatin sponge-wrapped anastomoses lags slightly behind that observed when this substance was omitted. The tables in the previous study show that even the slight narrowing obtained here may be avoided. That this degree of constriction may be of significance is suggested by the fact that slight dilatation of the aorta distal to the anastomosis was observed (Fig. 4). The recent studies of Holman on the mechanism of the production of poststenotic dilatation and the possible clinical implications of this phenomenon suggest that even slight degrees of constriction should be avoided whenever possible.[4]

In none of the present experiments was bleeding from the suture line a problem. Under most circumstances, bleeding from an anastomosis will respond to gentle compression or may require an additional suture or two. If the bleeding or ooze should issue from an area that is inaccessible for sutures after completion of the anastomosis, there is no contraindication to buttressing the vessel in that location with gelatin sponge. This may be undertaken in either the child or the adult, and the wrapping of a vascular suture line in an adult with gelatin sponge should have no ill effects. However, the present studies would indicate that it is probably desirable to avoid surrounding a pediatric vascular anastomosis with gelatin sponge whenever possible.

The persistence of the fine chromic (gut) sutures in readily identifiable form in the aorta for periods of well over a year corroborates a similar observation made previously in this laboratory.§ While this is at variance with currently accepted opinion concerning the rate of absorbability of this substance, more rapid absorption may be anticipated in

§ References 3 and 5.

other organ systems, such as the gastrointestinal tract.

Assimilation of the absorbable gelatin sponge in these puppies was apparently more rapid and more complete than in previous studies with adult animals from this laboratory.[1] Presumably the critical factor is not the age of the animals, but the fact that in the latter group the gelatin sponge immediately became saturated with blood, serving as a matrix for the organization of a connective tissue sheath.

SUMMARY

The abdominal aorta of a small series of puppies was divided, united with simple interrupted sutures of 00000 medium chromic absorbable gut, and the anastomoses wrapped with absorbable gelatin sponge (Gelfoam).

Growth studies were carried out during prolonged survival periods.

An increase in the diameter of the anastomosis was obtained consistently.

The rate of growth was slightly less than that observed in a control series where gelatin sponge had been omitted.

Postanastomotic dilatation was observed in the gelatin sponge series, in contrast to the controls.

When employed to reinforce pediatric vascular suture lines, gelatin sponge should be placed as a patch over a particular site rather than wrapped around the entire anastomosis.

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Skeletal Lesions in Coccidioidomycosis

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Coccidioidomycosis, for several decades recognized as an endemic infectious disease in the southwestern portion of the United States, is being recognized as a worldwide affliction. Its spread in this country is due primarily to contraction of the disease by members of the Armed Forces while stationed in the endemic area during World War II. Bone and joint involvement during the disseminated stage of the disease is the exception, rather than the rule. Its occasional appearance, however, demands that the orthopaedic surgeon be sufficiently familiar with its manifestations and behavior to recognize the condition when he is confronted wth it.

Coccidioidomycosis is a deep mycotic infection caused by the fungus Coccidioides immitis. The initial, or primary, phase is usually a self-limited pulmonary infection.* This may be a comparatively innocuous, subclinical condition. It is sometimes accompanied by chills, fever, malaise, headache, and multiple joint pains. Symptoms and signs of pleural involvement are frequently observed, but many patients develop no clinical symptoms. After an interval of two to six weeks lesions may appear in other organs, indicating the onset of the secondary or disseminated phase of disseminated coccidioidal granuloma.

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From the Orthopedic Department, Wadsworth General Hospital, Veterans Administration Center, and the Department of Surgery, University of California School of Medicine at Los Angeles.

* References 18 and 3.

C. E. Smith,[18] in his comprehensive review, states that the organism exists in two forms. In the saprophytic, resting phase, the chlamydospores, which may be grown on solid media, are square, rectangular, or eliptical, from 2 to 10μ in length, and 2 to 5μ in diameter. The better-known spherules constitute the parasitic form. These develop doubly refractile walls, and range from 10 to 60μ in diameter. The adult spherule ruptures, dispersing endospores, which, in turn, develop into young spherules. In the resting form the chlamydospores survive for years in soil subjected to wide temperature fluctuations but, in general, dry.

PATHOGENESIS

The portal of entry is almost always the respiratory tract. There may be occasional entrance through abrasions of the skin. The digestive juices are believed to destroy the spores. The inhaled chlamydospores rapidly assume the spheroidal form within the host. The incubation period is from 10 to 40 days. Clinically and by x-ray, the primary phase of the disease greatly resembles pneumonia with hilar adenopathy, except that it usually clears up in about three weeks. Within a week or two after the onset of symptoms allergic manifestations develop, and the skin test becomes positive. Usually some minimal pulmonary scarring remains after the primary phase has resolved. This may be cavitation, calcification, pleural effusion, or a small area of fibrosis. With the acquisition of sensitivity, as evidenced by the positive skin test, approximately 20% of the patients develop erythema nodosum or erythema multiforme.

The granulomatous phase is the result of a dissemination of the pulmonary infection. It is believed by Smith to occur in less than 0.2% of cases. The spread is endogenous by

way of blood stream and lymphatics and may involve almost any organ or tissue. It is usually manifest within a few months of the primary infection but occasionally delays its appearance until after the lapse of several years. Immunity is conferred by a single infection and is generally thought to be permanent. Animals can be infected. The disease has been reported in cattle, sheep, dogs, and rodents. In the laboratory, rabbits, mice, and guinea pigs have been infected. No instance of direct transmission from man to man, man to animal, or animal to animal are recorded. The sexes are affected equally. The erythematous manifestations are twice as common in females, whereas the granulomatous dissemination is five to seven times commoner in males. In members of the white race the mortality of the disseminated infection is somewhat less than 50%. The death rate among Negroes is 23 times that of whites and among Filipinos 180 times that of whites. The endemic areas are southern California, Arizona, New Mexico, Texas, Nevada, Utah, northern Mexico, Venezuela, and the Chaco area of Argentina.

PATHOLOGY

The reaction of the host to coccidioidomycosis is similar to that in tuberculosis, except that spherules are encountered. There is first an acute, purulent phase in which the inflammatory reaction is primarily leucocytic. This is followed by a chronic granulomatous reaction in which monocytes, epithelioid cells, and giant cells are observed. Necrosis and caseation occur. Unless the spherules are identified, one cannot differentiate the lesion of coccidioidomycosis from tuberculosis histologically. The two diseases are occasionally found concomitantly.[8]

HISTORICAL BACKGROUND

Wernicke [21] reported the initial case of coccidioidal granuloma, originating in Argentina in 1892. Rixford,[14] in 1894, described the first case in this country. Many subsequent accounts of coccidioidal granuloma have appeared in the literature. General interest in this disease was aroused by the classic studies of Gifford,† in 1936, and the subsequent reports of Dickson [4] and Dickson and Gifford.[5] Since then instances of its appearance in many countries have been documented. Forbus and Bestebreurtj,[7] in their excellent review in 1946, cite instances of its appearance reported in the southeastern United States by Lynch in 1920, in Chicago by Hirsch in 1923, in Brazil by Gomez and de Assumpsão in 1924, and in Italy by Radaelli in 1930, and of reports from China, Mexico, and Nicaragua in 1931, Louisiana in 1934, Hawaii in 1935, and Kansas in 1936. They aver that 6,000 cases were clinically recognized in the Armed Forces of the United States in all the quarters of the globe during World War II. They analyze 95 cases, 50 of which were fatal. Of these 95, all but 12 patients almost certainly contracted the disease while stationed in the known endemic area of southwestern United States.

BONE AND JOINT INVOLVEMENT

As stated above, the disseminated phase of the disease occurs in a small proportion of cases. Both its frequency and its mortality are greater in the darker-skinned males. The frequency of bone and joint involvement is not known but is obviously comparatively rare. Only muscle and the gastrointestinal tract are more infrequently invaded by hematogenous or lymphatic seeding.

A review of the literature disclosed reports of 118 cases manifesting skeletal lesions as follows:

Carter [2] in 1934	70
McMaster and Gilfillan [12] in 1939	24
Rosenberg et al.[15] in 1942	1
Goren [10] in 1946	1
Sashin [16] in 1946	2
Lamphier [11] in 1948	1
Miller and Birsner [13] in 1949	17
Fraser et al.[8] in 1951	1
Walker, O. R., and Hall, R. H.[20] in 1954	1
Thiemeyer, J. S., Jr.[19] in 1954	1
TOTAL	119

†Gifford, M. A.: San Joaquin Fever, Annual Report to Kern County (Calif.) Health Department, 1936.

Dr. R. W. Huntington, of Bakersfield, Calif., believes that skeletal lesions are relatively more common in children than adults.

X-RAY CHANGES

The roentgen changes seen in coccidioidomycosis have been described in detail by Carter.‡ Benninghoven and Miller,[1] and Fraser and his co-workers.[8] Several types of x-ray changes are enumerated. In our experience roentgenological manifestations, like the pathological changes throughout the body generally, resemble those found in tuberculosis. Regional atrophy is the initial finding. The lesions are primarily destructive. There may be periosteal proliferation with cortical expansion and sclerosis. Sometimes cortical erosion produces the irregularities maldescribed as cystlike. The cortex may be perforated. Rarely sequestra are observed. Pathological fracture is an infrequent finding.

The bone lesions exhibit a predilection for the bony prominences—tibial tubercles, malleoli, patella, trochanter, ends of clavicle, olecranon, and epicondyles. Involvement of practically every bone and of all the joints but shoulder and temporomandibular has been observed.

Bony lesions commonly commence near the ends of the long bones (epiphysis, metaphysis, or diaphysis). Their spread is not significantly retarded by the epiphyseal plate. Extension into adjacent joints sometimes occurs. Erosion of joint cortices may be observed. Soft tissue abscesses are usual. Joint lesions may be primarily synovial or subcortical. These may extend to bone or soft tissues. Multiple bone lesions are encountered more often than single lesions.

ASSOCIATED LESIONS

Active pulmonary lesions are infrequently present. More commonly evidence of old, now quiescent, pulmonary involvement is discerned. Cavitation, pleural thickening, and parenchymal cyst formation, sometimes with calcification, are not unusual. The mediastinal and retroperitoneal nodes are often involved.

‡ References 2 and 3.

Erythema nodosum is common and twice as frequent in women as men, as stated above.[1] This may be accompanied by a generalized purulent pustular eruption.[10]

Practically all organs except the gastrointestinal tract may be invaded. The symptoms and signs will vary according to the organs involved.

DIAGNOSIS

Diagnosis of coccidioidomycosis can be made positively only upon identification of the organisms. Typically, there is a history of the patient's having been in an endemic area, the southwestern portion of the United States, perhaps for only a few hours. From one to four weeks later symptoms and signs of pulmonary disease develop. Characteristically, but not always, the respiratory infection clears up spontaneously in a few weeks. During this period the allergic reaction occurs, and the skin test becomes positive.

Development of the granulomatous, or disseminated, phase of the disease cannot be distinctly separated from the primary phase. The granulomatous spread may become manifest by the development of abscesses in practically any organ or system. It is usually the soft tissue abscess, developed by extension from the bone lesion, which calls attention to the osseous infection. The roentgen changes observed in bone, simulating as they do those seen in tuberculosis, are not diagnostic. The bony lesions may also mimic the changes of blastomycosis, metastatic carcinoma, or osteomyelitis.

A positive skin test is fairly reliable, but only six weeks or longer after infection. A negative skin test early, followed by a positive skin test later, is conclusive. Increase of the sedimentation rate is of little value diagnostically but of some consequence in determining the activity of the disease over a period of time.

Eosinophilia of variable degree is present in approximately 20% of cases. In meningeal infection the spinal fluid may show a paretic type colloidal gold curve.

Demonstration of the spherules in pus or by biopsy of a lesion establishes the diagnosis. The organisms can be cultured. They may

Clinical Data in Cases Studied

Age	Race	Presenting Symptoms, Duration	Associated Lesions	Bones and Joints Involved	Diagnosis Made by	Therapy	Time in Hospital	Follow-Up	
29 (Case 1)	N	Headache, 1 mo.; crick in neck, progressive paralysis diagnosed as cord tumor	Retropharyngeal abscess, mediastinal nodes	C5	Vertebral body biopsy	Rx tracheotomy and drainage abscess Crutchfield tongs	28 days	Rapid sepsis; paralysis	Died resp. paralysis
36 (Case 2)	W	Malaise, weakness, chills and fever, 2½ mo.	Cavitation right apex; infiltration RUL; cerv. glands	D11, C4, skull	Culture of sputum (Dr. C. E. Smith laboratory)	Prodigiosin	4 mo. course; developed multiple abscesses	Gradual downhill	Died
50 (Case 3)	W	Malaise, weakness, fever—10 days	Thickening of RUL and RLL, mediastinal nodes cleared in one mo.; erythema nodosum	Left tibia and fibula; ankle (B), astragalus, cuboid, os calcis, carpal navicular 1 yr. later	Culture of sputum (Dr. C. E. Smith laboratory)	Left below knee amputation; prodigiosin	1 mo. and 8 mo.	Ankle drained off and on 19 mo. after onset; 2 yr. later no recurrence; good walker	.....
31 (Case 4)	N	Pain left hip 3 mo.; lump left groin 4 mo., and left side neck 1 mo.; RUI inf. 5 mo. previously	Abscess left ant. chest wall; abscess over left clavicle and left flank; cerv. nodes	Left greater trochanter; left clavicle and scapula	Aspiration abscess, left clavicle	Tracheotomy, prodigiosin	6 mo.	Gradual downhill course	Died
29 (Case 5)	N	Chills, fever, vomiting; pain left ear and upper arm, 12 days	Diabetes; abscess left foot; pleural effusion; many subsequent abscesses	Left great toe, right knee, right elbow, right foot	Sputum showed spherules	Prodigiosin	6 mo.	Downhill course	Died
53 (Case 6)	W	Low grade fever 3 yr.; scaly dermatitis 1 yr.; joint pains; abscess right tibia 2 mo.	Old apical scarring; profuse drainage right leg, left thigh; scaly dermatitis	Right tibia; left femur (path. fract.)	Pus from tibia showed spherules	Right leg drained; left thigh drained; path. fract.; amputation through fract. site; hip disarticulation	2 mo. and 11 mo.	1st amputations as life-saving measure; 2d for continued drainage; 2 yr. after discharge doing well; no drainage; gaining weight	Died—coronary disease 4 yr. after amputation
51 (Case 7)	N	Chest symp. 6 mo.; several subsequent abscesses chest wall 3-4 mo.	Numerous abscesses; lesion left midlung; mediastinal glands	Right clavicle and sternoclavicular joint; right elbow?	Aspiration right clavicular abscess	Prodigiosin	30 days	Rapid downhill course	Died
36 (Case 8)	W	Mass right foot 6 mo. (URI inf. 1 yr. ago cleared in 3 wk.); nodules of all fingers	Lesion LUL; abscess right shoulder; erythema nodosum	Left acromium; D11-12; right 4th metatarsal; left 2d rib	Pus right foot showed spherules	Prodigiosin; drainage of abscesses	6 mo.	5½ yr. after discharge intermittent swelling; right foot no drainage for 2 yr.; limitation motion and occasional pain right shoulder; foot and back get tired when mountain climbing sev. days for deer	.....
32 (Case 9)	N	Recurrence RUI (original RUI 9 mo. ago) diagnosis made in Army; discharged to V. A.	Mult. abscesses; mediastinal nodes	Right ileum and ischium; right 2-5-11 ribs; left 2-3 ribs; C 6-7; left scapula	Spherules in sputum	No specific therapy	8 mo.	Downhill course	Died

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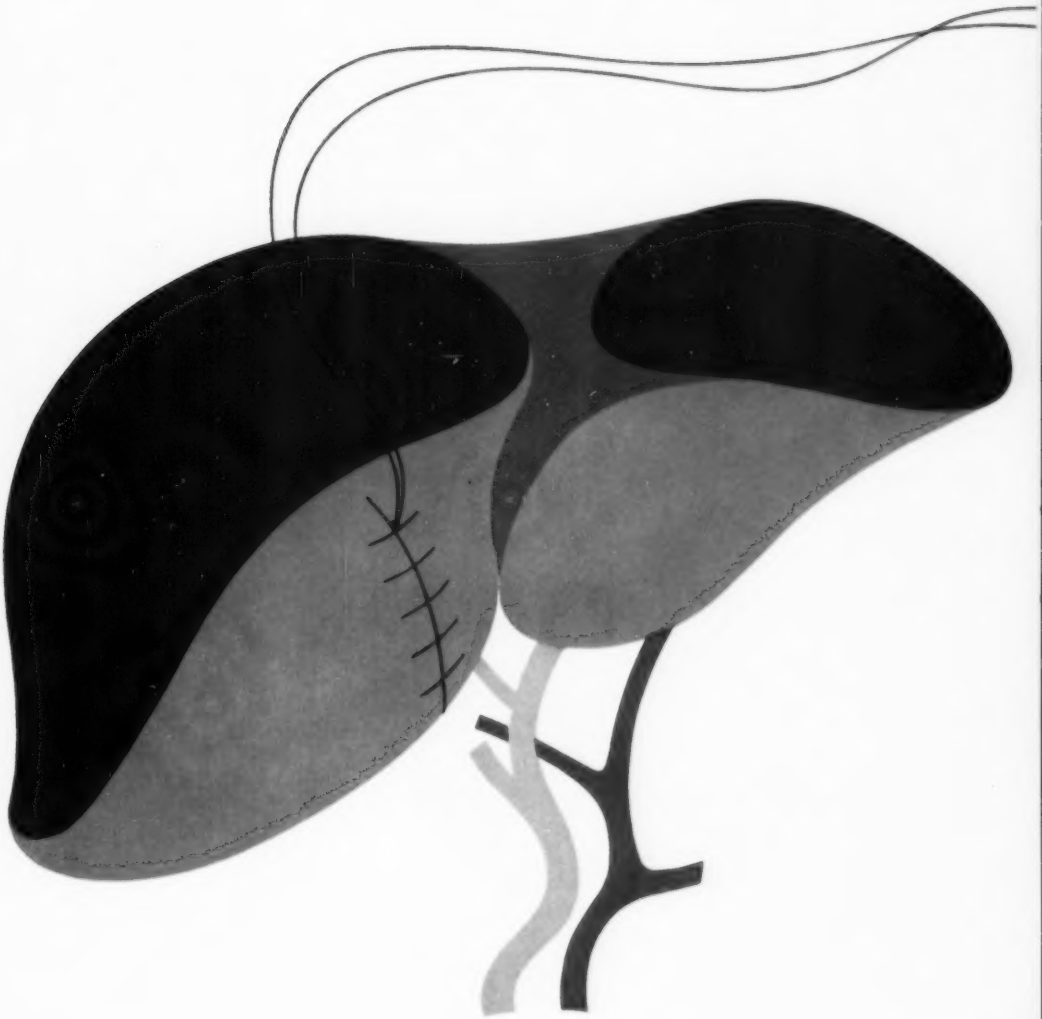

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27 N (Case 10)	Malaise; night sweats; cough; loss 20 lb.; mass over left hip 3 mo.	RUL lesion; sinus from hip abscess	Ischium, ileum	Pus from abscess left hip showed spherules	Drainage of abscess	6 mo.	3 yr. after discharge no recurrence—arrested; but still coughs	.....
21 N (Case 11)	Weakness and numbness; atrophy left median nerve 5 mo.; frontal headache 3 wk.	Headache, blurred vision, brain abscess	T1	Vertebral biopsy	.....	3 mo.	Downhill course	Died
24 N (Case 12)	Malaise, weakness, fever, 10 days	Mass right chest; temporal abscess, rt. right leg and foot	Right ileum; frontal bone; T9; L4-5; left scapula; left sacroiliac area; sacrum	Pus from temporal abscess	Temporal abscess cultured	28 mo.	Developed multiple abscesses; downhill course	Died
29 W (Case 13)	Swelling and pain left side neck 6 mo.	Left apical infiltration; matted cerv. nodes; block at T 3	C 5-6; L 3; T 5	Culture of drainage from cervical nodes	Prodigiosin, fungicidin (Nystatin) laminectomy	26 mo. at intervals	Still in hospital	.....
38 W (Case 14)	RLQ pain 9 mo.; mass Petit's triangle, 1 mo.	Psoas abscess communicating with colon and exterior; scarring LLL; retroperitoneal nodes	L 2-3; left femur; left ileum	Drainage and biopsy psoas abscess	Drainage psoas abscess; ileocolectomy; fungicidin (Nystatin)	4 mo. and 2½ yr.; still in hospital	Drainage from large indolent abscess involving gluteal muscles femur and ileum for 28 mo.; this closed after 8 wk. of Nystatin therapy and has been closed for 3 mo.	.....
34 N (Case 15)	Chest and abdominal pain; cough—sputum; loss 11 lb. 2 mo.	Left hilar and base lesions; mass rt. neck; general lymph adenopathy; pleural effusion, left	T 7; L 2-5	Sputum culture and aspiration neck abscess	Aspiration neck abscess; drainage left chest abscess	14 mo.	Gradual downhill course	Died
42 Fil (Case 16)	Recurrent pain, swelling left wrist 6 yr.; 3 op. prior to admission	Diabetes; fibrosis RUL	Left radius, distal end	Excision and biopsy mass left wrist, healed	7 local excisions of left wrist involving radius and ext. poll. long. and brev. tendons	Off and on for 11 yr.	Readmitted recently for wrist fusion	.....
55 M (Case 17)	Mass left ankle 1943 drained; astragalectomy 1945	None	Left ankle, left temporal area	Pus left ankle, biopsy left ankle	Amputation refused 1948	Off and on 7 yr.	Gradual downhill course	Died
23 N (Case 18)	Known cocci. for 1 yr.; pain neck and shoulders; drowsiness 2 wk.	Supraclavicular abscess; paralysis BUE, weakness RLE	C 4 to T 3 granuloma involv. meninges	Laminectomy tissue examination	Laminectomy C 4 to T 3	1 yr.	Developed extension of meningitis	Died
47 W (Case 19)	"Boil" dorsum left foot—1 mo.	None	1st metatarsal cuneiform jt.	Aspiration and culture; open biopsy	Curettement diethylstilbestrol (Stilbesterol)	15 mo.	Wound closed and remained closed 1 yr. after discharge	.....
33 N (Case 20)	Acute pneumonitis 1 mo.	L deltoid abscess; hilar adenopathy; apical fibrosis	R 11 rib	Aspiration and culture	Fungicidin (Nystatin)	still hospitalized	.....	.....
33 W (Case 21)	Pain, disability, synovial thickening and 15° motion right knee 10 yr.	Old hilar scarring	R knee	Tissue examination	Synovectomy	still hospitalized	.....	.....
32 N (Case 22)	Chest pain, cough night sweats, fever, weight loss 2 wk.	Many subcutaneous abscesses	Left clavicle, left ileum, left tibia	Sputum culture	Drainage of abscesses; fungicidin (Nystatin)	still hospitalized	.....	.....

be recovered from gastric washings, sputum, pleural or spinal fluid, and pus.§

The precipitin titer rises with the primary infection and within a few months disappears regardless of the clinical course.

The complement fixation may rise in low titer during the primary disease and remain for a variable period. In the disseminating case it continues to rise. A titer of 1:32 or greater is generally indicative of dissemination and a poor prognostic sign. In the patients who recover the titer will decline and perhaps eventually disappear after several years. (Chart).

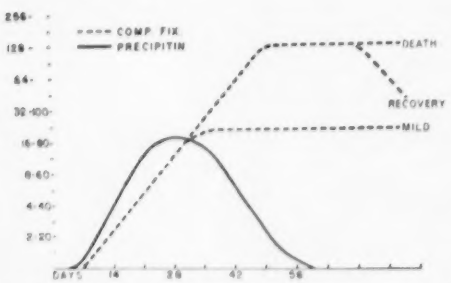


Chart showing complement-fixation titers in patients.

DIFFERENTIAL DIAGNOSIS OF BONE COCCIDIOIDAL LESIONS FROM TUBERCULOSIS

Carter [2] lists the following criteria as helpful in differentiating between these infections of bone:

1. Tuberculosis does not have a predilection for bony prominences.
2. Tuberculosis does not often have skin manifestations.
3. Tuberculosis is more frequently found in joints alone, which is unusual in coccidioidomycosis.
4. Tuberculosis generally causes less destruction of juxta-articular bone.
5. Tuberculosis is more likely to show destruction of nonopposing cartilagenous surfaces, coccidioidomycosis, of opposing cartilagenous surfaces.
6. Tuberculosis of joints usually takes many months to develop, while coccidioidal infections may occur much more rapidly.

TREATMENT

No specific therapy is known. The use of prodigiosin at this hospital for the past five years has not given conclusive results and is still being investigated. A regimen similar to that used in the treatment of tuberculosis is adhered to (except that streptomycin and the more recent nicotinic acid derivatives have no specificity for Coccidioides immitis). Surgery is utilized when indicated, as in any other granulomatous infection.

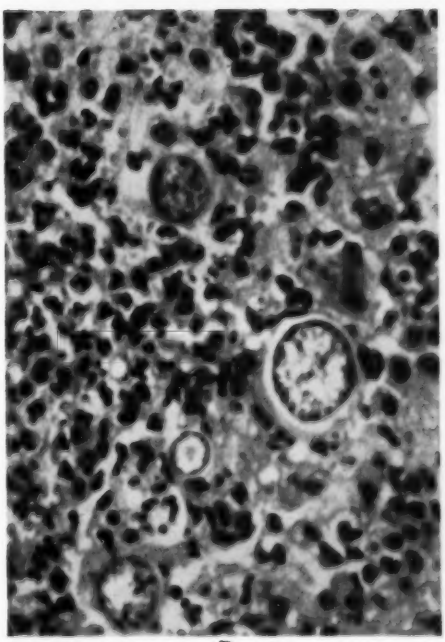
REVIEW OF CASES

During the past six years, 20 cases of coccidioidal infection of the skeletal system have been observed. Two additional cases from the Veterans Administration Hospital, Long Beach, have also been reviewed. All patients were adult males. All gave definite history of having spent some time in an endemic area. The ages varied from 21 to 55 years, the average being 31.45 years. Thirteen were Negroes, of whom 10 died. One was a Filipino. Eight were Caucasians, of whom one died. The percentage of deaths among Negroes is 77, in the entire group 50, among the Caucasians 12.5. All patients exhibited some degree of fever and elevation of the sedimentation rate. In six there was eosinophilia. Two of the 22 patients were also diabetic (Cases 5 and 16). In three instances death resulted from involvement of the brain or spinal cord (Cases 1, 11, and 18). In one case a massive granuloma involving the meninges and vertebrae from C4 to D3 was partly removed, but the patient died (Case 18). The course of the disease in the fatal cases was usually rapid. In two instances the patients were hospitalized for only 1 month; in one instance for 28 months, and in another, intermittently, for 11 years. The average (excluding the 11-year case) was 8.3 months. This is a little longer than the time given by most authors. On two occasions the diagnosis was established by a vertebral-body needle biopsy.

In two cases amputation as a life-saving procedure was done (Cases 3 and 6). One was done through the site of a pathological fracture of the femur with a surrounding abscess. Both patients are doing well two years after amputation. Neither now has any draining lesion. In another case amputation

§ References 6 and 18.

A



B

was advised but refused. This patient later died (Case 17).

The skeletal involvement was as follows:

Skull	4
Cervical vertebra	4
Dorsal vertebra	5
Lumbar vertebra	4
Clavicle	3
Scapula	3
Ribs	3
Carpus	1
Pelvis	5
Femur	3
Tibia and fibula	3
Tarsus	4
Sternoclavicular joint	1
Elbow	2
Wrist	1
Knee	2
Ankle	2
Small bones and joints of feet	2

Associated lesions observed in this series of patients, as one would expect, included, either from the history or during hospitalization, pulmonary involvement in all but five instances. Three of these were cases of coccidioidal meningitis in which the signs of old pulmonary involvement probably were overlooked. Two cases occurred in patients whose infections were of 7 and 11 years' duration. Enlargement of mediastinal or cervical nodes was noted in six instances. Erythema nodosum was observed in two patients. Abscesses in diverse areas were observed. Details as to age of patients, skeletal areas involved, associated lesions, and type of therapy instituted, etc., are shown in the Table.

REPORT OF CASES

The course of the disease varied greatly in these 22 cases.

The fulminating type of dissemination is illustrated by Case 1.

Fig. 1.—*A* shows the marked destruction of C5 (reproduced by permission from Fraser, C. G.; Monroe, S. E., and O'Hare, J. E.: *Annals of Surgery*, J. B. Lippincott Company, Philadelphia, July, 1951). *B* shows several typical spherules seen in the biopsy specimen (reproduced by permission from Fraser, C. G.; Monroe, S. E., and O'Hare, J. E., *Annals of Surgery*, J. B. Lippincott Company, July, 1951).

Fig. 2.—X-rays show (left) erosion of bodies of L4, L5; (middle) demineralization, thinning of cortex, and pathological fracture of left femur; (right) periosteal proliferation of right tibia, and thinning of fibular cortex.

A 29-year-old Negro laborer complained of headache for one month, followed in a few days by a crick in the neck and gradually increasing paralysis. He was thought to have a spinal cord tumor and was transferred to this hospital. X-rays revealed a destructive lesion of C5. Vertebral-body biopsy disclosed the spherules of coccidioidomycosis. Drainage of a retropharyngeal abscess, tracheotomy, and insertion of Crutchfield tongs failed to halt the rapid spread of the disease. He died 28 days after admission.

Figure 1*A* shows the marked destruction of C5. Figure 1*B* shows several typical spherules seen in the biopsy specimen.

An instance of chronic low-grade infection of several bones, with eventual healing, is depicted by Case 6.

A 53-year-old, white rancher was admitted on Nov. 19, 1949. He had a history of intermittent low-grade fever, malaise, and multiple joint pains for four years, with a loss of 90 lb. (40.8 kg.). Sixteen months prior to admission, an abscess of the right tibia was drained, and coccidioidomycosis identified from the pus. Three months prior to admission drainage from the left femur developed; he fell and sustained a pathological fracture of this bone. He was treated elsewhere and transferred to us in a spica.

His general condition was poor. There was profuse drainage from the left thigh. X-rays disclosed lesions of the left femur, right tibia and fibula, and of L4 and 5. On Nov. 29, 1949, amputation through the left thigh was performed. The wound was not closed. Copious drainage persisted. On Feb. 9, 1950, hip disarticulation was done. Eight months later, the wound had healed. Two years after surgery, the patient was working on his ranch, had gained 50 lb. (22.7 kg.) was in good general health, and had had no recurrence of drainage.

Figure 2 shows (left) erosion of the bodies of L4 and 5; (middle) demineralization, thinning of cortex, and pathological fracture of left femur; (right) periosteal proliferation of right tibia and thinning of fibular cortex.

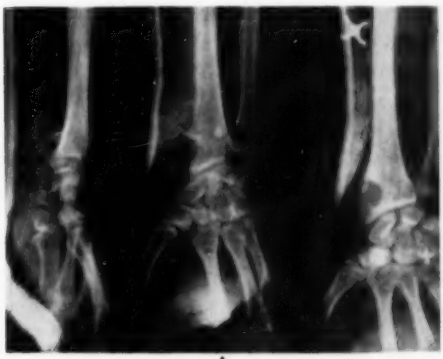
The possibility of infection with minimum exposure is illustrated by Case 12.

A 24-year-old Negro carpenter drove through the San Joaquin Valley in January, 1946. A month later he was hospitalized elsewhere for an upper respiratory infection. Shortly thereafter a right

Fig. 3.—*A*, photograph showing an abscess of the chest wall involving ribs and sternum. *B*, photograph showing an abscess of the left thigh involving the greater trochanter.

A

B



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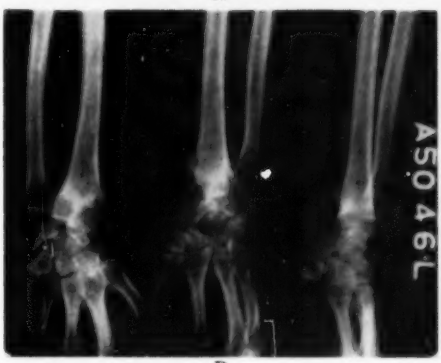

B

Fig. 4.—*A*, roentgenogram showing the destructive and productive lesion in the lower radius, taken in 1946, two years before his fourth hospitalization. *B*, roentgenogram showing recurrence of the lesion eight years later (Feb. 1, 1954), prior to wrist fusion. Involvement of wrist joint is evident.

temporal mass appeared. Drainage of the abscess revealed coccidioidomycosis. Numerous other abscesses then developed. He was transferred to this hospital six months after the onset of his illness. Many bones showed lytic lesions, and there was continuous drainage from multiple abscesses. He died 28 months after the onset of his pulmonary symptoms.

Figure *3A* shows an abscess of the chest wall, involving ribs and sternum.

Figure *3B* shows an abscess of the left thigh, which involved the greater trochanter.

The presenting symptoms and course of the disease in Case 14 were somewhat bizarre.

A 38-year-old white machinist was admitted to the hospital in May, 1951, complaining of right lower-quadrant pain of four months' duration and low-grade fever. He developed a transient, nonspecific prostatitis. No explanation for the right lower-quadrant pain and fever was discovered. He was discharged after three months' observation. One month later he was readmitted with the same complaints. At this time a tender mass was discovered in Petit's triangle. Incision and drainage of a right psoas abscess, which involved the bodies of L2-3, revealed coccidioidomycosis spherules. A fistula subsequently developed between the psoas abscess and the large bowel, necessitating iliocolostomy and, later, partial colectomy. Drainage from the wounds persisted. There has been an insidious extension of the infection locally. Twenty-six months after his first admission, the abscess had invaded most of the gluteal musculature, the upper femur and ilium. His general condition remained almost stationary, for months. Recently, on fungicidin (Mystatin) therapy, all abscesses healed; he gained weight and strength, and left the hospital.

An unusual occurrence of a single lesion persisting for 11 years without identification of any other focus is illustrated in Case 16.

A 42-year-old Filipino, a mild diabetic, was admitted on Oct. 27, 1948, complaining of a painful swelling of the left wrist for two weeks. He stated that he had had a similar lesion in 1942, accompanied by chills and fever. The lesion was drained, recurred, and was drained a second, then a third time. On admission he presented a frank abscess over the dorsum of the radial side of the left wrist, with tenosynovitis of the extensor pollicis longus and brevis tendons. Biopsy revealed a granulomatous lesion simulating tuberculosis. Two months after admission excision of the mass, including tendon

Fig. 5.—Roentgenogram showing the circumscribed punched-out lesions of right ischium and left ileum.

sheaths, and curettage of the distal end of the radius revealed typical spherules. The wound healed readily. The mass recurred and was again removed 18 months later. A third similar operation was necessary after another 18-month period. He was readmitted to the hospital for the seventh time on March 24, 1954. Astonishingly enough there was remarkably little limitation of function of hand or wrist and no evidence of any other lesion in the body. Wrist fusion was performed March 30, 1954.

Figure 4*A* shows the destructive and productive lesion in the lower radius; this roentgenogram was taken in 1946, four years after the original admission.

Figure 4*B* shows recurrence of the lesion eight years later (Feb. 1, 1954). At this time there is evidence of involvement of the wrist joint.

Roentgen lesions suggestive of multiple myeloma were found in Case 10.

A 27-year-old Negro student was admitted to the hospital on March 6, 1950, complaining of a swelling of the left hip, which had been present for three months, with rather rapid increase in size. There was a loss of 20 lb. (9.5 kg.), night sweats, and a productive cough. He had been in San Joaquin valley in the summer of 1945, and approximately one month later there had been an upper respiratory infection, which cleared up spontaneously. Six days after admission the abscess in the region of the left hip was incised and drained. From this, coccidioidomycosis spherules were recovered. Films showed a lytic lesion in the upper portion of the left ilium and right ischium and pulmonary fibrosis. Examination of the sputum failed to reveal any spherules. The abscess in the region of the left hip closed 11 months after incision. Since this date there has been no evidence of activity in this area. The patient was last seen two and one-half years after the operation, at which time his general health was good. There have been no recurrences of symptoms in the region of the left hip. Both bone lesions remained quiescent. The patient still had a chronic cough.

Figure 5 shows circumscribed punched-out lesions of the left ilium and the right ischium.

A more leisurely and benign course is exemplified by Case 3.

A 50-year-old white retired Army Colonel entered the hospital in December, 1948, complaining of malaise, weakness, fever, and much productive sputum for 10 days. Signs and x-ray changes in the right lung led to suspicion of a bronchogenic carcinoma or tuberculosis. There was an 11% eosinophilia and a sedimentation rate of 35 mm. The skin test was negative for coccidioidomycosis. Sputum culture (Dr. C. E. Smith's laboratory) established the diagnosis. All chest signs cleared within one month. He was discharged at the end of six weeks.

One month later he was readmitted with erythema nodosum and granulomatous ulcers on the chin, above the right external malleolus, and on the left great toe. Biopsy of the chin lesion revealed typical spherules. Three months later he was discharged with all lesions healed.

Eleven months after his first admission he developed a right epididymitis. X-rays revealed patchy areas of destruction of the left lower tibia and fibula, right cuboid, and talus. Twelve months after his first admission he was again readmitted because of ulceration in the region of both ankles. X-rays showed involvement of both ankle joints and the left os calcis. A month later the right carponavicular joint was also partially destroyed.

Nineteen months after his first admission, because of continuous profuse drainage in the region of the left ankle, a below-knee amputation was performed. Two years after his discharge from the hospital he was a good walker on his prosthesis. There had been no drainage from any area for 18 months.

SUMMARY

Twenty-two cases of coccidioidomycosis involving the skeletal system are reviewed. Invasion of a single bone or joint is unusual. No bone or joint is immune from invasion. There is no differentiating pathognomonic, clinical, or roentgen finding, although the last frequently simulates that of tuberculosis. The diagnosis is made by identification of the organism. Since practically any organ of the body may be involved, the clinical course of the disease follows no definite pattern. Dissemination may be rapid and fulminating, particularly in other than white patients. No specific therapy is known.

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Prostatic Cancer X

Comparison of Open and "Punch" Biopsy Techniques

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With the progressive increase in life span of the average American man, there has been a concomitant increase in the clinical incidence of and the death rate from adenocarcinoma of the prostate gland.[1] This increasing death rate from prostatic cancer seems surprising when one considers that a curative operation for early prostatic cancer was described almost 50 years ago.[2] Moreover, the cancer-free survival of properly selected prostatic cancer patients subjected to radical perineal prostatectomy compares favorably with curative surgery for adenocarcinoma arising in other parts of the body.[3] The increasing prostatic cancer death rate must then be attributed to the well known fact that even today approximately 95% of all prostatic cancers are first diagnosed clinically in metastatic phase.[4] Although promising palliative methods, notably castration and the administration of estrogenic substances, have altered the prognosis for the metastatically involved patient somewhat, such an individual lives only 36 months, on the average, following establishment of the diagnosis of metastatic disease.[5] Earlier diagnosis appears to be the only logical means by which the increasing cancer death rate can be altered.

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There are no symptoms which are typical of early prostatic cancer. It is also apparent that rectal palpation appraisal of the prostate gland is inadequate in itself as a means of detecting the majority of early malignant growths in the prostate gland.* Tissue diagnosis has become the reasonable recourse to which urologists may turn for reliable information concerning the nature of prostatic enlargement.

Since there are objections to the prostatic secretion smear technique (exfoliative cytology), attention is therefore directed to the surgical methods of obtaining tissue for diagnostic purposes.† Both the punch and the open surgical perineal prostatic biopsy techniques use specimens from the posterior prostatic lamella.‡ Approximately 85% of all prostatic cancers arise from this portion of the gland.§ A comparison of the two methods for the detection of early cancer seems warranted.

CLINICAL STUDIES

The main body of clinical material is made up of persons from the Bowery Series who did not seek medical advice or assistance spontaneously.[6] These 200 patients are persons in whom the diagnosis of early prostatic cancer was sought without any clinical indication that the patient might harbor a malignant growth of the prostate gland. In other words, these patients are in essence a survey of a segment of the aging male population for early prostatic cancer. The second group, consisting of 13 patients, were referred to the urological service of the Francis Delafield Hospital after a clinical suspicion of malignancy had been established elsewhere. For

* References 6 and 7.

† References 8 and 9.

‡ References 10 and 11.

§ References 12 to 15.

purposes of early cancer detection, the first group of patients are those to whom the greater significance is attached.

Each patient in both study groups is evaluated by means of medical history, complete physical examination, urinalysis, complete blood count, and blood chemistry studies, including nonprotein nitrogen, total protein, and serum electrolytes. In addition, a radiographic survey of the skeletal system and the thorax is made, and serum acid and alkaline phosphatase determinations included. Intravenous urography and cystoscopic examinations are also performed. A final clinical impression of the presence or absence of prostatic cancer is established immediately prior to performance of the combined biopsy.

TECHNIQUE

Under suitable anesthesia,[16] the patient is placed in exaggerated lithotomy position and a No. 24 F. prostatic urethral tractor is introduced and opened. After sterile preparation and draping of the perineal skin, a 0.5-cm. skin incision is made just lateral to the median raphe at a point one-third the distance from the anus to the urethral bulb. The prostate gland is then palpated against the prostatic tractor by the palmar surface of the left index finger. The modified Gusberg cervical punch instrument[10] is then introduced through the small skin incision and is guided by the palpating finger in the rectum to the posterior surface of the prostate gland. The cups of the biopsy instrument are opened as the tip enters the posterior anatomical capsule and a core of tissue is removed.

Immediately after performance of the punch biopsy, an open surgical dissection of the perineum is performed by a method similar to that of Belt.[17] The entire posterior aspect of the prostate gland is thus exposed, and a wedge of tissue extending the breadth of the prostate gland is removed at a point approximately midway between the apex and the base of this structure. The width of the wedge on its posterior aspect is from 0.5 to 1.0 cm. in order to facilitate longitudinal division of the biopsy specimens for both frozen and permanent sections.

A comparison between the surgical pathological diagnoses established upon tissue specimens from these two sources constitutes the main purpose of this report.

RESULTS

Biopsies were evaluated on the basis of the pathological findings in the following manner:

(*A*) If the specimen of prostatic tissue obtained by the punch method yielded the same diagnosis as that derived from microscopic study of the sample removed by open biopsy, the punch method was designated as adequate.

(*B*) If the prostatic specimen taken by punch biopsy was proved by microscopic diagnosis of the sample removed by the open biopsy technique to be incorrect, it was termed erroneous.

(*C*) If the tissue obtained was of other than prostatic origin or if the examination revealed it insufficient for diagnostic purposes, or if more than one of several samples were considered inadequate for diagnosis, the biopsy was termed unsatisfactory.

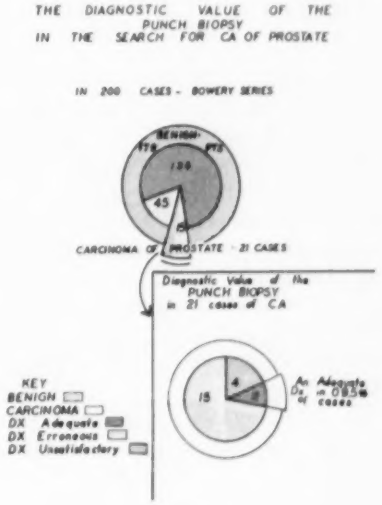

Figure 1.

Of the total of 200 consecutive Bowery Series survey cases, 21 were subsequently proved to have carcinoma and 179, benign prostatic tissue.

Of the 179 final diagnoses of benign prostatic tissue in the Bowery Series, the punch specimen was (1) adequate in 134 cases, or 69% of the total; (2) false positive in no instances; (3) erroneous in no instances; (4) inadequate in 45 instances.

In the 21 patients of the Bowery Series in whom the final diagnosis was carcinoma

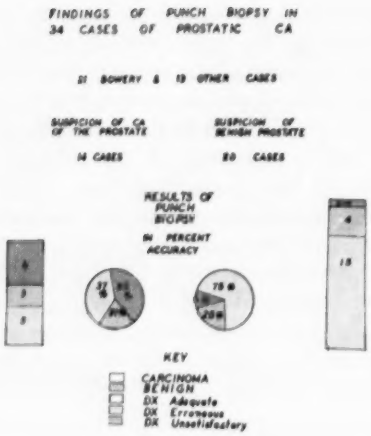

Figure 2.

of the prostate, the punch specimen was (1) adequate for diagnosis in 2 cases; (2) unsatisfactory for diagnosis in 4 cases; (3) subsequently established as being erroneous in 15 cases.

In the 13 patients of the non-Bowery Series in whom the final diagnosis was carcinoma of the prostate, the punch specimen was (1) adequate for diagnosis in 4 cases; (2) unsatisfactory for diagnosis in 3 cases; (3) subsequently established as being erroneous in 6 cases.

The value of punch biopsy of the prostate in a search for prostatic cancer among these 200 cases is shown graphically in Figure 1.

The results from punch biopsy are considerably better when there was a preoperative

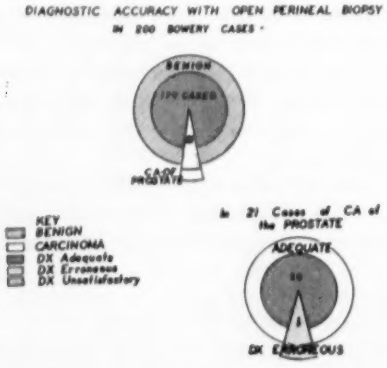

Figure 3.

suspicion or diagnosis of prostatic cancer. The results of punch biopsy testing in the 34 instances of cancer (viz., 21 in the Bowery Series and 13 general admissions), comparing clinical suspicion of carcinoma with final microscopic diagnosis, are shown in Figure 2.

The correct diagnosis of benignity was established by the open perineal prostatic biopsy technique in all 179 cases. The numerical correlation value of the open perineal biopsy method for benign prostatic tissue and the final diagnosis was thus +1.0.

Of the 21 cases of the Bowery Series which proved to be carcinoma, the perineal biopsy specimen was adequate and correct in 20 in-

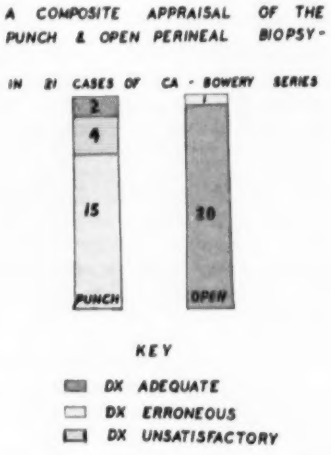

Figure 4.

stances. The numerical correlation value between the diagnosis obtained by open perineal biopsy and the final diagnosis was +.952. The total diagnostic value of the perineal prostatic biopsy technique is shown in Figure 3.

A composite appraisal of the punch and perineal surgical specimens as diagnostic methods for detecting early prostatic cancer is shown in Figure 4.

COMMENT

The obviously lower value of the punch method for detecting unsuspected carcinoma as elucidated by the data presented in 200

consecutive cases is perhaps explainable upon two bases: (1) the inability to direct the punch instrument with consistently good accuracy, and (2) the smaller amount of tissue, including posterior anatomical capsule, which is present in the punch specimen when compared with that obtained by the open technique.

When the punch method is used for documentation of cases of suspected or clinically diagnosed prostatic cancer, the accuracy of the method—and therefore its absolute value—is greatly increased. For this purpose it seems to be a worthwhile addition to the armamentarium of the urologist. This is especially true if future statistics upon the survival rates for prostatic cancer are to be based upon tissue diagnoses rather than clinical impressions, the latter containing in almost any series of large size a considerable number of false positive impressions of cancer.

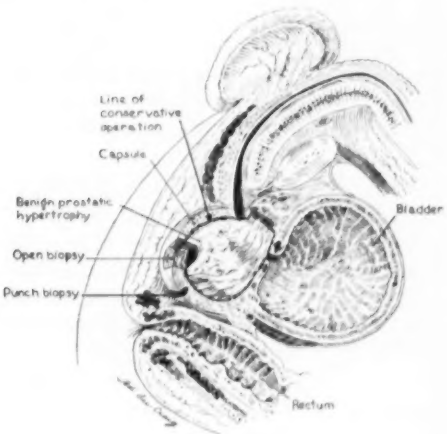

Figure 5.

Reports of enthusiasm for the punch technique published by other workers ‖ seem unwarranted when one considers the lack of suitable tissue (posterior lamella) with which the punch biopsy could be compared. The surgical pathological specimen acquired by any conservative prostatectomy, including transurethral resection, does not include posterior prostatic tissue (Fig. 5). Therefore, a negative microscopic impression of prostatic cancer based solely upon punch and ordinary prostatectomy specimens is inconclusive. Prostatic cancers can and do exist in the unsampled, posterior portion of the prostate. Such undetected malignant disease undoubtedly accounts for the significant incidence of prostatic cancer reported within 5 to 8 years following conservative prostatectomy.[19]

These several considerations have been reemphasized by the results of our comparative study of open and punch posterior prostatic biopsies.

‖ References 10 and 18.

SUMMARY

A comparison has been made between the values of punch biopsy and open perineal surgical biopsy of the prostate gland.

The punch biopsy has been shown to be of little value in the detection of prostatic cancer in a survey group of 200 consecutive patients.

The diagnosis made as a result of examination of tissue obtained by open perineal prostatic biopsy has shown a positive correlation with the final pathological diagnosis of +.995 in 200 cases.

The punch biopsy is considered of no value whenever the diagnosis from punch specimens does not reveal carcinoma.

The punch biopsy is of greater value in documenting known or suspected cases of prostatic cancer than it is for the detection of early, curable lesions.

For the purpose of detecting prostatic cancer in its curable stage, open surgical biopsy of the prostate through the perineal anatomical approach is superior to other methods.

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Diabetic Neuropathic Arthropathy of Feet

Summary Report of Seventeen Cases

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In neuropathic arthropathies of the feet there is a history of one or more of the following conditions: diabetes, syphilis, nerve and spinal defects, birth defects (idiopathic myelodysplasia), trauma, alcoholism, syringomyelia, leprosy, poliomyelitis, and peripheral nerve disorders. The diabetic form of neuropathic arthropathies of the feet has been brought to the attention of the general practitioner, the internist, and the orthopedic surgeon with increasing frequency. Whereas tabes of syphilitic origin was formerly the usual cause of these painless deformities of the feet, with complicating soft tissue infections, ulcers, and osteomyelitis, the diabetic neuropathic foot is now showing the higher incidence. Perhaps there is greater alertness in recognition. Certainly there must be a correlation with the higher incidence of diabetes generally, coupled with poor public awareness of the possible consequences of neglect.

At the same time that reports on this diabetic foot disability are appearing more frequently, there have been noticeably fewer reports of tabetic, nutritional, and chemical neuropathic arthropathies. Here public education in the control of syphilis and improved mass nutrition may explain this fortunate decline. Neuropathic arthropathies resulting from injuries to nerves and cord during World War II have also shown an increase, but our concern in this article is with the growing diabetic complication. The present study comprises a series of 17 cases seen over a five-year period.

From the Cook County Hospital and the Chicago Medical School.

Read before the Section on Orthopedic Surgery at the 103rd Annual Meeting of the American Medical Association, San Francisco, June 22, 1954.

SUMMARY REPORT OF SEVENTEEN CASES

Inquiry into the history in 17 cases of diabetic neuropathic arthropathy of the feet, seen over a five-year period, revealed that 16 patients failed to follow the diabetic diet, refused treatment of the diabetes entirely, or were careless in cooperating with its medical management. Insulin had been prescribed in 16 of the cases, but only 10 of these patients showed any attempt to follow instructions. In the one well-controlled case of diabetes, deformity did nevertheless occur.

TABLE 1.—*Classifications of Neuropathic Arthropathies of the Feet (Five-Year Period*)*

Etiology	Cases
Diabetes	17
Tabes	4
Spinal dysraphia	4
Idiopathic	2
Alcoholic	1
Traumatic cord	1
Leprosy *	2

* U. S. P. H. S., Dr. A. Mayoral's cases.

Swelling of the foot, sweat gland dysfunction, with excessive perspiration early in the condition and absence of perspiration later, eczematoid lesions of the skin, and, in a few cases, waxy appearance of the skin indicated autonomic nervous system involvement. The peripheral pulses, oscillometric readings, and subpapillary venous plexus filling time were within normal range in all cases. In several cases thermocouple readings (U. M. A. machine) were normal. In most cases the toes and the plantar surface of the foot were warm and pink.

Fig. 1.—Bilateral diabetic foot deformity. Note "rocker sole" of right foot.

A typical deformity of the pseudotabes arthropathy (*pied tabétique*) can be seen in Figures 7 and 8. In three cases a peculiar deformity of the plantar surface with convexity, or "rocker sole," occurred. This was apparently due to the displacement of the midtarsal joint or portions of the tarsus (Figs. 1 and 2). In two cases this deformity caused small ulcerations at the plantar surface of the foot at approximately the tarsometatarsal joints. Destruction of an ankle joint (Figs. 5 and 6) occurred after surgical intervention for a fractured ankle joint. There was calcification of the small blood vessels in many instances.

An example of metatarsophalangeal joint involvement with local plantar ulcerations and toe deformity with overriding is seen in Figures 9 and 10. An unusual condition of compounding of the skin with "bursting"

Fig. 2.—Roentgenographic appearance of conditions shown in Figure 1.

Fig. 3.—The "bursting" compound ankle deformity, with wide gap of tissue separation at site of ankle joint.

effect on the tissues is seen in Figures 3 and 4; in this case there was involvement of both the tibioastragalar joint and the metatarsophalangeal joint. A great deal of bone destruction occurred in both areas, with a sudden compounding of the ankle joint immediately preceding a state of diabetic coma, with ensuing death.

TABLE 2.—*Summary of Joint Involvement in Seventeen Cases of Diabetic Arthropathic Feet**

Location	Number
Tibiotarsal joint	1
Tarsal	1
Tarsometatarsal (Lisfranc's joint)	9
Metatarsophalangeal	8
Interphalangeal	2

* There were several cases of multiple joint involvement.

The anatomic joint involvement in our series included the tibial tarsal joint and tarsal joint in one case each; the tarsometatarsal joint (Lisfranc's) in nine cases; the metatarsophalangeal joint in eight cases, and the interphalangeal joint in two cases. Multiple joint involvement occurred in several cases (Table 2).

Fig. 4.—"Bursting" compound ankle problem seen roentgenographically. Note also the involvement of metatarsophalangeal joint.

Fig. 5.—Ankle deformity. Poor operative result. Extreme displacement with destruction of tibiotarsal joint.

Fig. 7.—Typical deformity of diabetic arthropathy with pes planus, valgus forefoot, and short wide deformity.

DIFFERENTIAL DIAGNOSIS

Although the Charcot-like deformity of the foot is poorly understood, it is known that an affected nervous system is the immediate cause in the presence of a long-standing or neglected underlying disease, such as syphilis or diabetes. A history is often elicited of precipitating or initiating trauma in conjunction with the deformity.

Careful neurologic examination is essential to thorough diagnostic appraisal. Disturbances in sensation of the feet, diminished or absent reflexes, and reduced pupillary responses to light and accommodation are diagnostically indicative. It is likewise important to assay the peripheral circulation, which, curiously, in this condition is normal. A rule embracing the clinical diagnostic criteria may be formulated:

In the presence of diabetes mellitus, a painless foot with reduction or loss of vibratory and position sense and diminished reflexes, in which physiologic trauma produces a progressive deformity with abnormal widening and shortening, and flattening and eversion of the foot; in which there is good peripheral circulation, but roentgenologic evidence of bone displacement with hypertrophic or atrophic changes—such a combination of conditions is unequivocably pathognomonic of diabetic neuropathic arthropathy of the foot.

In advanced cases of gross deformity, osseous destruction in the involved joints and

Fig. 6.—Note destruction of lower end of tibia and fibia and early absorption of astragalus. Local bone overgrowth is also present.

Fig. 8.—Roentgenographic appearance of typical deformity shown in Figure 7. Note tarsometatarsal displacement (Lisfranc's joint) and bone displacement.

Fig. 9.—Severe overriding of toes, with deformity, ulcerations, and cellulitis.

Fig. 10.—Note metatarsophalangeal joint destruction, osseous absorption, and spindle-like metatarsal bones.

Fig. 11.—Shortening and widening of foot as a result of joint destruction.

various osseous defects are seen roentgenographically, with periosteal proliferation and combined radiolucency and sclerosis, along with soft tissue swelling and with ulceration or tissue necrosis.

Laboratory data are helpful in the treatment and prognosis but not essential to diagnosis. Chemical analysis of the blood may show the red cells reduced to 2,000,000, in which case the waxy pallor of the skin and mucous membranes will evidence the anemia on physical examination. The blood urea nitrogen may show a 26 to 77 mg. per cent increase per 100 cc. Fasting blood sugar will be greatly increased in untreated diabetic cases. Urinalyses will give data typical of diabetes. Analysis of the spinal fluid may show an elevation of protein content but ordinarily does not do so. Oscillometric readings vary from 2 to 8 units, which are normal values at the calf area.

Gangrene of an extremity or cellulitis, also found in diabetes mellitus, must be differentiated by involvement of the peripheral circulation, intense pain, tenderness, and difficulty in weight-bearing—none of which is symptomatic in diabetic neuropathic arthropathy of the foot. The significance in diagnosis of osteomyelitic destruction of bone and displacement of the various joints of the foot must also be evaluated. These, too, are associated with pain, extreme swelling, and evidence of sepsis.

The tabetic neuropathic arthropathy is found in tabes dorsalis with a positive Kahn or Wassermann reaction or positive spinal fluid findings. Unlike the tabetic arthropathic

Fig. 12.—Roentgenographic appearance (same case as shown in Fig. 11).

foot, the diabetic foot shows general bone atrophy, greater destruction of bones and joints, particularly in association with infections and soft tissue thickening, and cellulitis. The downward course of the diabetic foot deformity probably begins with either bilateral or unilateral foot thickening and degenerates progressively to flattening, eversion, and external rotation associated with destruction of the tarsus and fragmentation, small bone displacement, and, eventually, extensive disruption of the ankle, tibioastragalar joint, tarsometatarsal or metatarsophalangeal joints, or any combination of these. Ultimately there may occur osseous destruction and soft tissue lesions. Associated with the grave joint deformity and the basic diabetes there may finally develop gastrointestinal disorders, genitourinary disturbances (particularly bladder paralysis), and involvement of the autonomic nervous system.

A parenthetical observation may be worth incidental watching: It is unlikely that in bilateral cases the deformities begin in the two feet simultaneously, but these cases are not seen early enough to be certain. The question of laterality is open to observation. One foot is usually worse than the other in bilateral cases. In our series, in one bilateral case the onset was known to have been in the left foot, and there were four other left foot involvements, and eight right.

TREATMENT

The complex condition discussed in this paper requires supervisory medical control of the diabetes, painstaking care of associated complications, and unrelenting foot hygiene. Deformity may develop in various forms, necessitating surgical excision of displaced bones or joints, which may otherwise cause ulcers, and the treatment for associated infections, ulcers, or osteomyelitis. Local amputation may be necessary for improvement.

Supportive medical treatment must be instituted with the administration, as advisable, of one or more of the following: vitamin B_1 and B_{12}, dilute hydrochloric acid, crude liver extract, and injections of pregnant mammalian liver extract (Biohepulin). Vasodilators may be indicated. Antibiotics are prescribed if infections are present.

Surgical procedures are often imperative and certainly worth trying. Table 3 lists the types of associated surgery done in our series of 17 cases, namely, incision and drainage for a local infection in a neuropathic foot, transmetatarsal amputation in another case, and in 3 cases, amputations of the toes necessitated by ulcers and soft tissue infections with cellulitis. Sympathectomy is sometimes advisable. Local amputation may give improvement, but total amputation is justified only in the extreme case in which general health necessitates it. Arthrodesis of the ankle or tarsus, or both, may be required if deformity is extreme. Care of osteomyelitis is imperative.

TABLE 3.—*Operative Procedures in Seventeen Cases of Diabetic Neuropathic Feet*

Type of Surgery	Cases
Incision and drainage	1
Transmetatarsal amputation	1
Toe amputations	3
Open reduction of ankle	1
Exostectomy	1
Nerve biopsy	2

In one case involving the tibial astragalar joint, an open reduction for a fracture of the medial malleolus resulted in complete disintegration of the ankle, absorption of the osseous and cartilaginous material, and tibiotarsal dislocation. Dorsal foot exostectomy was performed in one case of tarsal displacement and local ulcer formation on the plantar surface of the foot. A purposeful anterior tibial nerve removal was done in two cases, one, at the same time that a dorsal exostectomy was done (Figs. 11 and 12). Biopsies revealed the usual finding seen in such cases in the peripheral nerves, namely, degeneration of axis cylinders, loss of the myelin sheath, and increased connective tissue elements.

General nonoperative orthopedic care calls for physical therapy in the form of thermoregulated contrast baths and boric acid or soapsud soaks, special shoes, prostheses, arches, braces, crutches, and canes. Frequent and prolonged elevation of the affected foot or feet gives relief.

SUMMARY AND CONCLUSIONS

Seventeen cases of diabetic neuropathic feet, found in a five-year survey, have been observed; three of these were cases in private practice.

The increasing incidence of this deformity as is evidenced by the growing number of reports in the literature calls attention to another medical and surgical challenge in an age in which diabetes grows more prevalent and the average life span grows longer.

Physiologic trauma and neuropathy associated with diabetes mellitus are basic factors in the occurrence of such arthropathies of the feet.

With the associated neuropathy there are an accompanying anesthesia, paresthesia, loss of sensation in joints, loss of or diminished reflexes, pupillary changes, and reduced vibratory and position sense.

Nerve tissue studies have not commonly been reported in these cases. We were able to obtain sections for study in two cases. Biopsies revealed typical peripheral nerve degeneration with loss of the myelin sheath and increased connective tissue elements.

Ulcers, tissue necrosis, or nutritional tissue changes do not usually occur concomitantly with the progression of the deformity but are more likely to occur later after the deformity has taken place. On the other hand, in several cases, pressure necrosis or ulcers and low-grade tissue infections did appear earlier, which can probably be explained on a nutritional-tissue basis. In such cases there was roentgenographic evidence of advanced bone atrophy and bone changes.

Roentgenologic surveys in all cases disclosed two main types of osseous changes: (1) the atrophic type with dissolution of joints, absorption of bone, and atrophy of the metatarsus and tarsus; (2) the hypertrophic type with local destruction but with overgrowth of bone, and calcification in and around the involved joints. The second type occurred more frequently in our series. Displacement of bone, either in a dorsal or plantar direction, was not uncommon.

The vascular survey in all cases revealed satisfactory peripheral circulation.

The deformity apparently develops slowly and insidiously, without warning, without prodromata. There is often a history of accident, however, dating the patient's first awareness of disturbance or in retrospect appearing to be the time of onset. The usual end-result is first seen by the physician as a painless foot deformity with an exaggerated shortening and widening of the plantar surface and occasionally exhibiting a "rocker sole" as well as soft tissue degeneration.

Surgical intervention by exostectomy or fusion is indicated only if deformity is severe and weight-bearing difficult because of an acute problem of proper shoe-fitting. Local amputation or excision may improve the condition.

In most instances neglect is revealed in the management of the diabetic problem. On the other hand, even in the presence of good diabetic control, the complicating deformity may result; nevertheless, the underlying disease will make the complication more hazardous if it is not carefully controlled as a primary effort in over-all correct treatment.

Further requisites of treatment are supportive measures, such as the administration of the vitamin B complex, adequate nutrition, and antibiotics to combat infection.

The associated problems in our series were not unusual but might have been expected in a group whose average age was 53.17. The complications were primarily hypertension and heart disease as well as obesity. There were in addition instances of glaucoma, rheumatoid arthritis, cancer, cellulitis, alcoholism and cirrhosis of the liver, syphilis, and gangrene.

There was one death which occurred during diabetic coma.

In general, diabetic neuropathic arthropathies of the feet are grave, often leading to total disability, with reduction or even loss of gainful occupation. Treatment is empirical and conservative, and the prognosis must be guarded.

6 N. Michigan Ave.

Extrahepatic Biliary Duct Obstruction Due to Stricture

Changes in Morphology and Physiology of the Liver and an Attempt at Prognosis

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The purpose of this paper is to review the experimental and clinical facts pertinent to obstruction of the external bile ducts, with special reference to strictures, and to present the clinical, laboratory, and pathologic features of a collected group of cases in an attempt to formulate some basis for prognosis and treatment.

PREVIOUS WORK

How soon do clinical signs of extrahepatic obstruction appear in the experimental animal? In dogs, bile appears in the urine in 24 to 28 hours; scleral staining in 5 to 10 days, and generalized tissue icterus in 9 to 26 days after division and ligation of the common bile duct with the gall bladder intact.[1] General signs of illness appear in the dog as anorexia and apathy at four weeks. As early as two days, there is an abrupt rise in total cholesterol and esters which is maintained for four to six weeks and then falls gradually to near or below preoperative levels. There is a lowering of albumin and elevation of globulin so that by eight weeks there is a reversal of the A/G ratio. The galactose tolerance test remains normal even up to 17 weeks. A significant elevation of alkaline phosphatase occurs by the second day, rises rapidly for 8 days and significantly for 5 weeks more, and then falls at 60 to 80 days. The rise in serum bilirubin occurs between the second and the sixth days, and continues gradually thereafter, maintaining a plateau with a late fall. There is no increase in the amount of indirect bilirubin. Bile appears in the urine by the fourth day. The concentration of fecal urobilinogen falls rapidly. A little persists in the feces. The persistence of urobilinogen in the feces indicates that excretion of bile occurs into the bowel lumen from the blood. This theory is supported grossly by the observation that the fecal urobilinogen associated with long-standing biliary obstruction is found in the thin shell of dark brown feces covering a core of acholic stool.[2] Prothrombin time remains normal up to three to four months, when the prothrombin content falls steadily but no hemorrhagic tendency appears. The thymol turbidity is not affected. All these changes occur in the absence of inflammatory changes in the liver.[2] These changes, as culled from the literature, are summarized in Figure 1.

After obstruction, the volume of the extrahepatic duct system increases, ultimately reaching enormous proportions. When the gall bladder is in communication with an obstructed ductal system, the bile in the ducts varies in color from dark green to black. Its consistency is often ropy. The color and consistency depend somewhat upon the duration of the obstruction. When the gall bladder is absent or separated from the ducts, the fluid within the ducts gradually becomes colorless, consisting almost entirely of the secretion of the duct walls.[3] It is called "white bile."

Hepatic ductal and parenchymatous morphologic changes occur slowly. The obstruction of the left hepatic duct of the cat or dog

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Lauren V. Ackerman, M.D. reviewed the pathological specimens and Dr. Carl A. Moyer helped in the preparation of this manuscript.

is attended by the continued secretion of bile into the left hepatic ductal system.[4] Four to six weeks after the institution of the obstruction, the main left hepatic ducts are grossly dilated. The dilatation is accompanied by varying degrees of atrophy of the ductal epithelium. The small ducts at the periphery of the portal spaces do not share in the dilatation. The lining cells of these ducts remain cuboidal. Their lumina are more or less obliterated, and their number per unit of slide surface is increased. According to Harley, this increase in number of small ducts per unit area of slide surface is due to growth in length and tortuosity of the ducts.[4]

After four to six months of extrahepatic ductal obstruction, richly nucleated fibrous tissue develops around the hepatic lobule but does not extend into them. The distribution of this connective tissue is irregular. There may be a marked decrease in the number of bile ducts. At 12 to 14 months, the fibrous tissue becomes more adult in type, with flattening of the nuclei; the bile ducts are greatly dilated, and there is marked decrease in the size of the individual lobule. A small amount of bile is constantly present outside the biliary passages in the periportal area of the lobule.[4] The liver cells, when compared with the unobstructed area, are unchanged. The glycogen content of the liver with extrahepatic obstruction is the same in all segments so long as dietary fat does not exceed one-third of the total caloric intake.[5]

In the experimental animal, the gross appearance of the liver is unchanged for four to six months. Then the lobular outline becomes more distinct. After 12 months of ductal obstruction, an irregular shrinkage of the liver occurs, particularly along the free edge, and the surface becomes irregularly roughened. The color of the liver is yellow during the first weeks of obstruction, but ultimately becomes dark green-brown.[4] A comparison of these observations in animals with the hepatic physiological and morphological alterations following obstruction of the flow through the extrahepatic biliary ductal system in man * are summarized in Table 1.

From the experiences of others listed in Table 2, it is clear that one of the most pressing problems related to surgery of the biliary tract is the elucidation of the factors which make a postoperative stricture of the com-

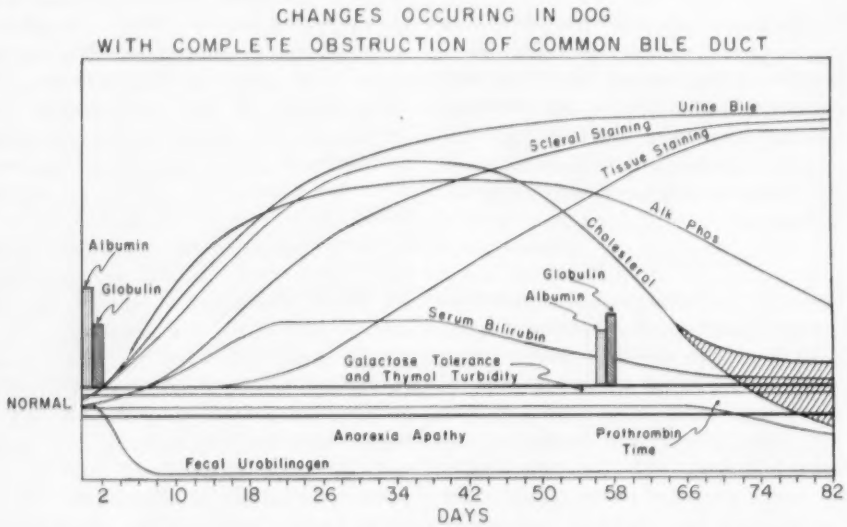


Fig. 1.—Changes occurring in dog with complete obstruction of the common bile duct.

References 6 through 11.

TABLE 1.—*Comparison of Morphologic Changes in Dog and Man Resulting from External Biliary Duct Obstruction*

Time After Obstruction	Morphologic Changes in Man	Morphologic Changes in the Dog
4 Days	Inflammatory cell infiltration "open-network" appearance of portal connective tissue Dilatation of bile ducts and changes in lining epithelium	.....
10 Days	Pronounced bile retention	.....
51 Days	"Proliferation" bile ducts and thickening of epithelium Progressive increase in connective tissue and inflammatory infiltrate	.....
4-6 Weeks	.....	Dilatation of large ducts and atrophy of lining cells Bile retention "Proliferation" of small ducts without dilatation
4-6 Mo.	.....	Progressive increase in richly nucleated fibrous tissue Small ducts may decrease in number
12-14 Mo.	.....	Marked fibrous tissue proliferation Marked dilatation of ducts Marked decrease in lobule size
General	Inconstant variations in glycogen content of lobule; regeneration of liver cells; small amount of free bile in periportal area	No change in glycogen content Regeneration of liver cells; small amount of free bile in periportal area

mon bile duct such a dire surgical complication from the standpoint of ultimate complete recovery. The high immediate postoperative death rate indicates that obstruction of bile ducts by stricture produces profound metabolic changes about which there is little experimental evidence. Are they related to alterations in hepatic function and other factors?

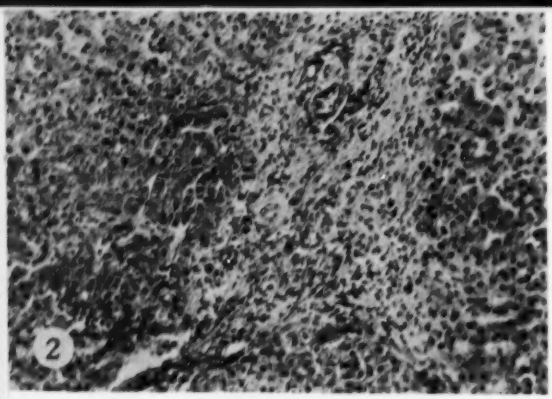
MATERIAL AND METHODS

A correlation was attempted of the so-called tests of liver function with the hepatic morphologic changes and clinical course of 12 patients with stricture of the external biliary ducts who were operated upon in Barnes Hospital between 1945 and 1953. Observations included preoperative concentration of protein, bile pigments, alkaline phosphatase or euglobulin (cephalin flocculation) in the serum or plasma, the retention of sulfobromophthalein (Bromsulphalein®), the fasting blood sugar, or the prothrombin time (Table 3). One of the patients in this group known to be living has recently recovered from an episode of recurrent cholangitis. Liver biopsy of this patient (Patient 175332) shows marked fibrosis and parenchymal distortion but normal-appearing parenchymal liver cells in the presence of completely normal "liver function tests" after six years of intermittent obstruction with chills, fever, and jaundice (Fig. 2). In the past, the "liver function" tests performed upon this

TABLE 2.—*Results of Operative Correction of Strictures*

Author	Cases, No.	Operative Deaths, No.	Postop. Deaths, No.	½ Yr.	1 Yr.	2 Yr.	3 Yr.	4 Yr.	5 Yr.	8	10	12	15	20	30
Eliot [12]	109	24	15	8	25	18	12	10	20	7	10	6	2	4	1
Walters [13]	98 ('24-'39)	10	18	53% had good results at five years											
	65 ('40-'48)	2	10	37% had good results at one year											
Cole [14]	63	4	..	42 (71%) good to excellent results											
Douglas [15]	12	3	2	..	1	..	3	2	3	..	..	..	..	..	..
McArthur [16]	5	0	0	1	..	1	..	..	3	..	..	..	..	..	..
Pearse [17]	106	..	..	80.1% good results											
	97	..	..	62% good results											
Whipple [18]	59	..	2	40 rehabilitated											
Lahey [19]	54 ('17-'39)	..	12	17—no symptoms 10—minimal symptoms*											
	185 ('40-'48)	..	12	50—no symptoms 88—minimal symptoms											

* 40% of these died during the next five year period.

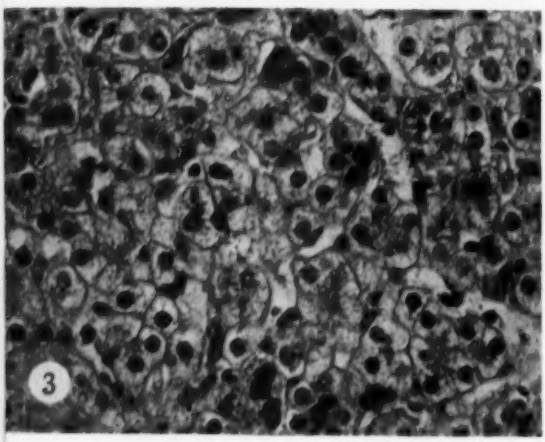


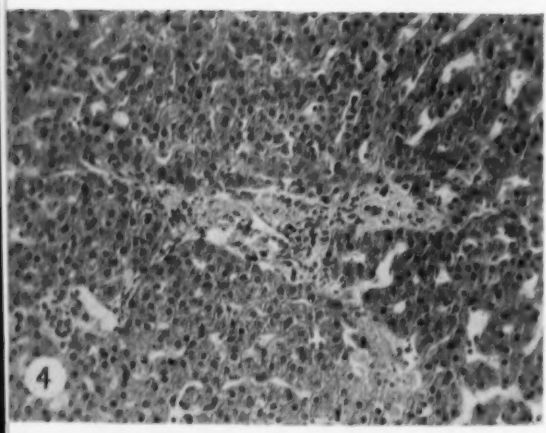
patient had been abnormal, often to extreme degrees, but without correlation with the microscopic changes demonstrated by concurrent liver biopsy.

In an effort to correlate the microscopic findings of liver biopsy with the clinical findings and subsequent course, sections of liver removed during the operation upon the ducts were reviewed microscopically without knowledge of the clinical course of the cases.

OBSERVATIONS

All sections of liver showed varying degrees of bile retention as evidenced by dilated collecting ducts, more or less filled with bile thrombi and similar thrombi in smaller radicals and canaliculi (Fig. 3). Bile was also seen frequently outside the ductal system, especially in the liver cells of the central lobular zone, free and within macrophages in the periportal area. Nothing was seen that could be interpreted as "reaction" to bile. Inflammatory cellular infiltration was confined mainly to two loci: (1) Leucocytes, lymphocytes, and macrophages were seen in small irregularly distributed focal areas of parenchymatous destruction, and (2) in the periportal areas, the infiltration containing fewer macrophages and more leucocytes and lymphocytes. Fibrosis of the portal area (Fig. 2) was present in varying degrees in all except one case (Fig. 4). The changes described by others [6] as "open network" could be identified in some cases, and fibroblasts of varying ages [4] were seen on serial sections. The fibrous tissue increment was definite, well organized, and, most frequently, adult in type. In some cases of long duration, there was an encroachment upon the parenchymal lobules by fibrous tissue, with distortion of lobular outline. The great number of small bile ducts in the periportal area was an outstanding feature. No edema was seen. The liver cells themselves showed no changes in





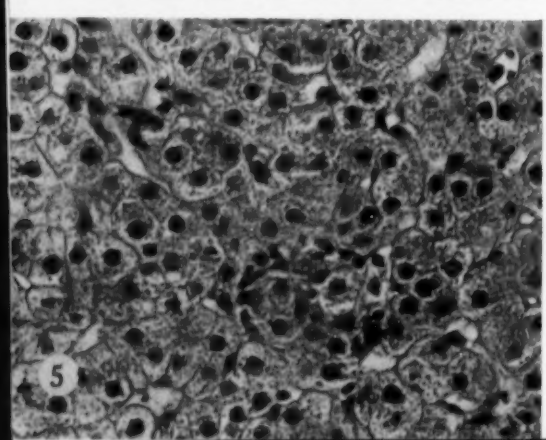


Fig. 2.—Note prominent fibrosis and parenchymal distortion but normal-appearing parenchymal cells after six years of intermittent obstruction.

Fig. 3.—Note bile retention with bile thrombi in small ducts and canaliculi.

Fig. 4.—Note absence of portal fibrosis in this case.

Fig. 5.—Photomicrograph to demonstrate no changes in parenchymal cells of the liver in a patient with longstanding intermittent obstruction.

structure or staining quality which might be interpreted as a generalized degenerative process (Fig. 5).

The intensity of the polymorphonuclear inflammatory reaction could not be correlated with the clinical evidence of infection (chills and fever). The livers of some persons who had suffered repeated bouts of chills and fever contained few leucocytes, and those of others who had never had chills or fever occasionally contained many. There appears to be no relationship between lymphocytic infiltration and the clinical findings.

So long as obstruction existed and there was increased bilirubin concentration or icterus index elevation, there was microscopic evidence of bile retention as shown by thrombi of biliary pigment in the collecting ducts and bile capillaries, especially in the central and peripheral portions of the lobules. The higher the concentration of bilirubin in the serum, the larger and more extensive were the thrombi. There was never more than a small amount of bile free in the periportal areas, and there was nothing that could be interpreted as a cellular "reaction" to bile.

The increase in number of small bile ducts per unit slide area was most pronounced in persons with prolonged or recurrent episodes of pain, chills, fever, anorexia, weakness, and jaundice. Relief of the obstruction did not allow resolution of the ductal "proliferation." The ductal proliferation remained long after the relief of the obstruction. However, the extent of ductal proliferation seemed to be ultimately limited and even reduced by the fibroplasia.

The development of fibrosis, particularly in the periportal area, was associated with bile retention and duct "proliferation." In general, it was most marked in persons having a long history of chills, fever, and jaundice. However, in some cases with a long history of chills, fever, and jaundice, there was little fibrosis (Fig. 5), while in the livers of many persons having had a short illness without chills or fever, much fibrosis was found. The amount of fibrosis was not relatable to the degree of icterus. However, the length of illness seemed important insofar as the type of fibrosis which developed. In those cases in which serial biopsies were available, the earlier sections showed a more loosely packed fibrous tissue element and the later sections a more compact fibrous tissue. However, at times, still later sections showed a more open fibrous tissue network than prior sections. Pruritus was not constantly associated with fibrosis.

Cellular degeneration and the inflammatory cellular infiltration in focal areas could not be correlated with the clinical course of the illness. The cellular degeneration occasionally seen with extrahepatic biliary duct obstruction due to stricture was not accompanied or followed by the hepatic cellular regeneration (mitoses and cell division) seen following the widespread hepatic regeneration attending hepatitis. However, the hepatocellular lobular reaction to obstruction was characterized by architectural irregularities of tortuosity of cords, variance in size of hepatic cells, and distortion of the usually uniform lobular outline and pattern. These differences in hepatocellular responses provide basis for microscopic differentiation of posthepatitic cirrhosis from biliary obstructive cirrhosis. The glycogen content of hepatic parenchymal cells appeared to be grossly normal in all sections studied.

COMMENT

There is disagreement as to the cause of the morphologic changes in the liver which accompany obstruction of the extrahepatic bile ducts. Some believe that the retention of bile is the etiologic factor and others that infection is of primary importance.† If fibrosis is due to a reaction to bile, why is none seen in the central lobular zone where bile pigment is most constantly found? If infection or bile retention are of primary importance, why is fibrosis so marked in congenital biliary atresia when no evidence of infection exists and very little bile retention occurs in the periportal areas. Bile salts are noxious substances: may they be the inflammatory agents? We do not know, but should

† References 7, 20, 21, and 22.

TABLE 3.—*Laboratory, Microscopic, and Clinical Findings and Duration*

Hospital Number and Age	"Liver Function" Tests													Microscopic Bile Retention	
	No. of Adm.	T. P.	Alb.	Glob.	Alk. P.	CCP	T. T.	L. I.	Bil.	FBS	BSP	NPN	P. T.	Fibrosis	Lobule	Duct
189960* 35	5th	7.6	3.9	3.7	10.5	4	5.6	15	18	..	....	..	77%	2+	0	+
169516 51	1st	7.8	3.9	3.9	33	0	3.0	40	..	81	....	21	....	0	0	+
157867 48	1st	6.8	3.9	2.9	22	1	13.8	..	4.4	..	22%	..	22%	1+	Autopsy 0	0
189960* 33	1st	8.6	4.5	4.1	28	3	5.5	26	1.66	60	....	19	60%	2+	0	+
123320 34	6th	6.0	3.8	2.2	12	2	4.5	..	2.8	..	80%	..	....	0	0	0
123320 34	7th	...	...	...	15.4	..	...	..	...	..	....	..	....	0	0	0
175332* 32	8th	8.2	4.8	3.4	144	3	6.6	..	6.09	..	....	10	83%	2+	+	0
175332* 32	9th	7.2	4.0	3.2	24	0	5.2	..	3.04	..	....	..	81%	2+	0	0
175332* 32	11th	6.0	3.4	2.6	56	3	...	21	2.37	73	....	12	50%	2+	1+	0
175332* 32	14th	6.9	3.6	3.3	..	..	...	..	4.46	..	....	..	100%	2+	0	0
188393 52	1st	7.0	4.2	2.8	..	2	3.8	6	11.8	15	....	32	70%	3+	0	0
72861 55	4th	8.5	4.4	4.1	..	..	...	..	50	..	....	..	80%	1+	0	0
118988 48	4th	6.5	3.9	2.6	18	1	...	98	2.26	117	....	20	70%	2+	1+	1+
123809 43	5th	6.9	3.5	3.4	32	3	...	120	..	100	....	15	60%	2+	1+	1+
121670 36	1st	6.6	3.8	2.8	45	0	...	71	..	..	....	13	73%	3+	1+	1+
149032 55	1st	7.2	4.2	3.0	..	..	2.1	83	..	.	....	22	100%	1+	0	0
220509 52	1st	8.4	3.5	4.9	39	3	14.8	23.4	..	.	....	..	100%	1+	±	0

* Indicates cases included more than once.

of Symptoms of Patients Studied at Barnes Hospital 1945-1953

Months of Duration of Symptoms and Clinical Appearance												
									Enlargement			
Duct Prolif.	Liver Cells	Jaundice	Chills	Fever	Weight Loss	Anorexia	Pruritus	Ill ?	Liver	Spleen	Ascites	Remarks
												Complicated with death
+	Norm	42	42	42	5	1	0	Chr	4FB	0	0	Shock in O. R. without recovery; died 1st p. o. day
0	Norm	36	0	0	0	0	0	Chr	6FB	0	?	Progressive uremia and liver failure; died one month post-op.
0	Norm	60	60	60	0	0	60	Chr	3FB	2FB	0	Hyperpyrexia and coma with death on 3d p. o. day; repair over T-tube 5 years prior
												Complicated with recovery
+	Norm	24	24	24	5	1	8	Chr	4FB	1FB	0	Tant. tube placed above stricture 12 mo. prior; finally recovered after severe hypotension and coma 3 days
												Uncomplicated with recovery
0	Norm	120	120	120	0	0	120	Acute RUQ	3FB	3FB	0	Cholecystectomy 1943 with cholangitis; re-op. 6 mo. later and stricture found; symptoms returned immediately post-op.; 1945 had choledochoduodectomy over T-tube; symptoms continued and with many admissions, re-op. 1952 and Roux-Y choledochojejunostomy done; symptoms returned in 6 months after last op.; well now for 3 mo.
0	Norm	126	126	126	0	0	126	0	2FB	0	0	
2+	Norm	42	42	42	20	2	42	Chr	3FB	0	0	Cholecystectomy 42 mo. prior with post-op. cholangitis, 2 more op's in 6 mo. without persistence of symptoms; op. 2½ mo. prior with inop. stricture; spontaneous external fistula with intermittent symptoms; on 8th admission had choledochoduodectomy; on 9th, stricture of anastomosis resected and duct-to-duct done; no symptoms until pulled T-tube out, then recurrent cholangitis; on 11th admission stricture resected and T-tube put in; intermittent cholangitis becoming more severe when pulled T-tube out—replaced immediately at op.
2+	Norm	48	48	48	0	0	48	Acute RUQ	0	0	0	
2+	Norm	56	56	56	20	1	56	Chr	3FB	0	0	
2+	Norm	66	66	66	0	0	66	0	0	0	0	
0	Norm	22	0	0	0	0	22	0	4FB	0	0	Repair 2 yr. prior unsuccessful; no icterus pre-op.; re-adm. 3 mo. later with proven bleeding varices; died 18 mo. after repair over T-tube; death due to bleeding varices
2+	Norm	42	42	42	16	1	0	Chr	1FB	0	0	Repair 2 yr. prior unsuccessful; recurrent cholangitis 5 yr.; re-op. twice in next 3 yr. without relief; progressive symptoms—and died
2+	Norm	14	14	14	5	3 days	14	0	0	0	0	Symptoms of intermittent obstruction continued; died 18 mo. of liver failure
2+	Norm	36	36	36	8	1	36	Chr	5FB	2FB	0	Two repairs over T-tube in 12 mo. prior; unsuccessful; improved; no recent follow-up
2+	Norm	5	0	0	18	1	4	0	8FB	1FB	0	Next 30 mo. had recurrent symptoms; pregnancy interrupted at 2 mo. because of poor health; recurrent symptoms for 5 yr.
1+	Norm	12	12	12	0	0	0	0	1FB	0	0	Had external fistula for 1 yr. pre-op.; recurrent symptoms until T-tube removed 13 mo. post-op.; minimal symptoms since
0	Norm	20	20	20	20	0	20	0	5FB	0	0	Continued to have intermittent symptoms until obstructed T-tube removed; cholecystectomy 1933; expl. 1951-1952 without relief; minimal symptoms

we assume that the intensity of itching is proportional to the concentration of bile salts, then we can say that they likely have no relationship to the hepatic morphologic changes seen, for pruritus is not constantly associated with any morphologic change (Table 3). Clinical infection as manifested by chills and fever is not constantly related to morphologic hepatic changes or is the duration of clinical icterus. Functional and microscopic morphologic evidences indicative of parenchymal liver cell damage are rarely found in association with strictures of the extrahepatic ducts. In this series, even those patients with so-called "liver-death" showed no morphologic change in the liver cells, and none of them showed preoperative hepatic impairment as judged by clinical function tests. However, it is obvious that functional damage to the liver follows common ductal obstruction; the high incidence of postoperative deaths and hepatic insufficiency bespeak it. Walters [13] reported 28 deaths, with 9 due to hemorrhage and 13 to hepatic insufficiency; Judd,[23] 4 deaths, with 3 due to hemorrhage and 1 to hepatic insufficiency, and Douglas,[18] 5 deaths with 1 due to hemorrhage and 2 to hepatic insufficiency. In this series, there were three deaths during the immediate postoperative period, all due to hepatic insufficiency and none due to hemorrhage. Lahey [19] reported 12 deaths between 1917 and 1939. Five were due to hemorrhage and two to hepatic failure. He reported 12 more between 1940 and 1948. However, the actual mortality dropped between 1939 and 1948: 1917-1939, 12 deaths in 48 cases; 1940-1948, 12 deaths in 184 cases.

What are the causes of the relatively high mortality still associated with operative correction of strictural obstruction? Hypoxia in the presence of abnormal hepatic function results in central lobular necrosis, and this, according to Ravdin, is the primary cause of death during anesthesia.[5] Hypoxia may result from inadequate oxygen content of the inhaled gases, inefficient oxygen exchange within the lungs, or interference with the blood supply to the liver. The prolonged periods of hypotension, the coma or delirium and the hyperpyrexia associated with hepatic failure are evidence of profound physiologic alterations attending parenchymatous liver cell damage. There is no evidence that fibrosis or duct proliferation per se has any influence on the parenchymal liver function. The bile salts retained during the periods of ductal obstruction are noxious, but their exact role is yet to be established.[24]

Stricture of the external biliary ductal system is due almost entirely to operative trauma.[25] Operative trauma is inversely proportional to the experience and skill of the surgeon.‡ Table 4 shows the location, cause,

TABLE 4.—*Cause, Age, Sex, and Area Involved in Cases of Benign Extrahepatic Biliary Duct Stricture*

Author and Reference	Cases, No.	Causes							Sex		Decade of Age						Stricture Area					
		Operation	Cholangitis	Fibrosis of Ampulla	Adhesions	Trauma	Stones	Others	Female	Male	3	4	5	6	7	8	Hepatic	Cystic	Common	Hepatic and Cystic	Common and Cystic	Junction
Lahey & Cattell *	250	208	11	24	1	2	..	4	191	59	11	26	46	33	6	1	77	..	116	..	18	25
Douglas [18]	12	12	..	..	..	..	..	..	9	3	2	3	7	..	..	..	6	..	6	..	..	..
McArthur [16]	5	4	..	..	..	..	1	..	4	1	..	..	..	..	..	..	1	..	2	..	2	..
Pears [17]	2	2	..	..	..	..	..	..	2	..	..	..	..	..	..	..	1	..	..	..	..	1
Walters [28]	80	77	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Cole [14]	49	32	11	..	..	..	..	6	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Eliot [26]	42	42	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	5	31	..	..	..
	3	2	1	..	..	..	..	..	2	1	..	2	1	..	..	..	3	..	..	..	..	..
	45	13	..	..	..	..	32	..	26	12	5	8	10	13	2	..	5	..	9	31	..	..
Eliot [18]	36	21	2	..	..	..	4	..	..	..	..	..	..	..	..	..	7	..	16	4	..	9
	524	413	28	24	1	2	37	10	234	76	18	39	64	46	8	1	105	5	180	35	20	35

* References 19 and 25.

‡ References 19 and 25.

age, and sex incidence of a collected group of cases.

Table 5 shows that if a stricture follows cholecystectomy, it often dooms that individual to repeated surgical procedures. The operative treatment of strictures has been manifold. Eisendrath [34] has shown very clearly that if the injury to the common duct

TABLE 5.—*Stricture Results in Multiple Operative Procedures*

Author	Number of Patients	Number of Operations
Lahey [19]	239	761
Whipple [18]	54	144
Eliot [35]	38	80
Walters [12]	67	212
Author	12	45
	410	1242

having no symptoms or with only minimal or decreasing frequency of obstructive symptoms. However, a paucity of symptoms even five years after initial repair does not insure continued health. Lahey [19] found that 40% of patients having little difficulty 5 years postoperatively were dead 10 years after initial correction.

In the present series of 12 cases, 5 are known dead, 3 probably dead, 1 unknown, and 3 well. However, all three known survivors have been operated upon within the past six months for recurrent cholangitis. The average survival time is 6.1 years, with a high of 10 years and a low of 4 years. The number of operative procedures ranged from two to eight with an average of 3.75. These

TABLE 6.—*Results of Surgical Repair of Stricture Considering Type of Repair Utilized*

Type of Repair	Cases, No.	Well	Recurrent Obstruction	Died in the Immediate Post-Op. Period	No Follow-Up or Died Out of Hospital
End-to-end or plastic	196	119	44	7	7
Dilatation and tube	41	30	2	1	..
Biliary intest. anastomosis	161	98	20	10	11
Tubes	203*	146	57	..	..
Fistula excision or implant	22	5	7	4	6
	626	398	140	22	24

* This includes Pearse's cases. References 13, 35, 17, 15, 18, and 19.

is recognized immediately and steps taken to correct it at that time, the mortality and morbidity is nil. Of 27 cases that had immediate (i. e., at same operation) correction of the surgical defect (by a total of 14 different methods), none developed stricture. Of 31 cases with operative injury to the bile ducts which were treated with delayed surgical repair (i. e., another operation but soon after), 7 are known to have developed stricture; 5 were dead from hemorrhage, liver abscess or hepatic failure; 3 had cholangitis or fistula; results were unknown in 3, and the remaining 13 were well. However, operative correction of biliary ductal stricture still remains as the only means of saving the lives of persons with stricture of the common duct. Table 2 shows the results of cases collected from the literature. Each author uses a different method of expressing his results, but in general, the "good results" are persons

patients averaged five admissions for their illness, with one having 14 admissions in four years.

Table 6 shows the results when they are considered in terms of the type of repair or anastomosis done. Pearse's [17] detailed cases were reported on a short term follow-up. Few share his enthusiasm for the Vitallium tube, and none has been able to duplicate his results. In explaining this, Pearse says, "Some physicians became over-enthusiastic and expected it to be a cure-all. Others have used it to bridge a gap and have been disappointed when failure occurred." [36] In his cases, 11% of the tubes became plugged. Walters and Cole,[37] in discussing the problem, indicate they have had a rather high incidence of plugging of Vitallium, rubber, or polythene tubes. In general, the importance does not lie in whether a duct-to-duct anastomosis or a duct-to-intestinal anastomosis is

made, though the former has the advantage of less infection, but rather in the fact that a mucosa-to-mucosa anastomosis must be made to insure success. Whenever the stricture is so situated or so extensive that an anastomosis between fibrous scar tissue with a lumen and duct or intestine must be made, the operation is doomed to failure. Recurrent stricture eventually occurs. The procedure of hepatoenterostomy reported by Longmire as a method of treatment when no ducts can be found may be life-saving. However, the reduction of over-all mortality from stricture primarily rests upon noninjury to the duct during cholecystectomy.

SUMMARY

In summary, analysis of 12 cases indicates that there is no single laboratory test, gross or microscopic liver alteration, or combination of these factors which permits the formulation of an estimate of the clinical course of the disease.

The one finding of value on which to base an estimate of the clinical course of the patient is the general bedside state of the individual. If a patient is judged "chronically ill," this finding indicates an unfavorable outcome. Chronically ill patients die either during the operative procedure, immediately following it, or soon thereafter of hepatic disease or one of its complications (Table 3).

Experimental and clinical data have established many known facts about the effect of biliary obstruction. Its actual occurrence precedes its clinical manifestation by varying periods due to the reservoir capacity of the extrahepatic biliary system, and this capacity depends greatly on the degree of obstruction.

No relationship was found between obstruction and infection and to the morphologic changes known as "biliary" or "obstructive cirrhoses." Certainly both are associated with the cirrhosis (clinically and microscopically) and after the relief of both there may occur varying degrees of regression of the pathologic features. It is likely that there is great interdependence of all the factors.

Efforts to formulate some correlation between morphology and the laboratory tests of "liver function" have failed. The clinical study of the patient remained as the single most significant finding in determining prognosis.

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Effect of Splenorenal Shunts on Estimated Hepatic Blood Flow

Measurement by Radioactive Colloid

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Surgical production of splenorenal shunts in the treatment of portal hypertension has stimulated interest in the dynamics of liver blood flow. The objective of such shunts is to eliminate bleeding from esophageal varices, presumbably by reducing portal vein pressure. The reduction in portal vein pressure achieved by such a shunt can be measured by direct manometric reading at the time of surgery. However, such data are of limited value. The measurements can be obtained only at the time of operation; they vary with the systemic blood pressure, and they are frequently not reproducible in closely adjacent areas of the same vein.

It would seem desirable, then, to have some additional measure of hepatic hemodynamics. Bradley and co-workers * have estimated the liver blood flow by sulfobromophthalein (BSP) clearance. They found a reduction in the hepatic blood flow of cirrhotics, which was further reduced after a portacaval shunt.

Dobson and Jones [4] have utilized the disappearance rate of a radioactive colloid cleared from the blood by the reticuloendothelial system to measure hepatic blood flow. The technique is simple and safe. In addition, the presence of bilirubinemia does not interfere with the determination.

This method was utilized to study the effect of splenorenal shunts on the hepatic blood flow of patients with cirrhosis and bleeding esophageal varices. The volume of blood flow through the cirrhotic liver varied little from the normal volume. Production of a splenorenal shunt usually resulted in a reduction of liver blood flow. This reduction seemed to have little correlation with portal vein pressures observed at the time of operation.

METHODS

Nine patients with well-established portal hypertension and cirrhosis of the liver were studied. Eight had suffered repeated blood loss from ruptured esophageal varices. One had a shunt performed prophylactically when splenectomy for hemolytic anemia was done. In all, biopsies of the liver provided definite evidence of cirrhosis and x-rays demonstrated the presence of varices.

Liver function studies varied widely, but all the patients had marked reductions in sulfobromophthalein clearance and A/G ratios. In all, the cephalin flocculation test suggested liver damage. A clinical summary of each patient with pertinent data is appended (Addendum).

In all the patients the spleen was removed and an end-to-side anastomosis was established between the splenic vein and the left renal vein by the suture technique.

The technique of liver flow determination was that described by Dobson and Jones.[4] Four microcuries of colloidal chromic phosphate containing P^{32} were injected into an antecubital vein. Heparinized blood samples were withdrawn from the opposite antecubital vein at 2-, 3-, 4-, 6-, 8-, and 10-minute intervals. Two cubic centimeters of blood were plated and assayed for radioactivity. The activity was plotted on semilogarithmic paper as a function of time, and the best straight line was fitted

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* References 1, 2, and 3.

to the points. The slope of this line is the disappearance constant or the fraction of the blood volume flowing through the liver per minute. Blood volume determinations were done by combining T-1824 blue dye with the chromic phosphate.

The determinations were done early in the morning, with the patient fasting. These were repeated in a similar fashion postoperatively, just prior to the patient's discharge from the hospital, when he was afebrile and ambulatory. In some cases duplicate determinations were done several days apart. These yielded identical data.

The validity of this method and the assumptions necessary for its use in estimating the liver blood flow in normal animals and humans have been discussed in two previous papers.† Of the assumptions discussed in these publications, the two that would be expected to be the most important in dealing with cirrhosis are, first, the efficiency of the liver for colloid removal and, second, the speed of mixing.

The minimum value for the blood flow through the liver (assuming perfect efficiency) is obtained by multiplying the blood volume by the disappearance constant k. The disappearance constant may be calculated from the half-time of the disappearance by the formula, $k = \frac{0.693}{t\,1/2}$. For calculation of the actual blood flow, the extraction efficiency must be known. Owing to the fact that the disappearance rates obtained in this group of cirrhotic patients are nearly normal, one is led to predict (in spite of the one low efficiency which is measured) that the efficiency for colloidal chromic phosphate removal is high in cirrhosis and should, therefore, present no problem in the interpretations of the observed results.

RESULTS

The results of these studies are summarized in the Table.

Effect of Splenorenal Anastomosis on Portal Pressure and Estimated Hepatic Blood Flow

	Preoperative			Postoperative					
							△ Portal	△ Estimated Hepatic Blood Flow	
Patient	Portal Pressure, Cm. Saline	Blood Volume, Cc.	Estimated Hepatic Blood Flow, Cc/Min.	Portal Pressure, Cm. Saline	Blood Volume, Cc.	Estimated Hepatic Blood Flow, Cc/Min.	Pressure, Cm. Saline	Cc/Min.	%
S. F.	39	6126	1838	32	4200	714	— 7	—1124	—61
C. R.	28	5790	1910	24	4082	898	— 4	—1012	—53
L. L.	27	7256	2757	30	6329	1327	+ 3	—1429	—52
C. G.	37	5177	1812	27	4513	1128	—10	— 684	—38
C. S.	25	6629	1854	29	5185	1400	+ 4	— 454	—24
R. B.	Not done	5430	1790	16	5202	1473	....	— 317	—18
T. G.	36	5400	972	41	5855	1054	+ 5	+ 82	+ 4
L. W.	24	6400	1856	20	6089	2074	— 4	+ 218	+12
H. B.	32	5159	1238	24	7452	1416	— 8	+ 178	+14

Bradley reports a decreased efficiency for sulfobromophthalein removal by the cirrhotic liver, which is still further reduced in the presence of a splenorenal shunt. A single efficiency measurement for chromic phosphate removal by a human cirrhotic liver was below normal (about 70% of normal). No measurements have been made, however, comparing preoperative and postoperative efficiencies. An increase in the removal efficiency after shunting would enhance the significance of the results reported here, in that a low efficiency will yield an abnormally low estimate of the hepatic blood flow. Thus the observed decrease in colloid removal rate would be subject to interpretation as a decrease in actual blood flow of even greater magnitude.

The mixing of the injected dose has been shown to be rapid in the normal human. The T-1824 injected simultaneously with the chromic phosphate is rapidly mixed.

† References 4 and 5.

These cirrhotic patients were found to have a preoperative estimated hepatic blood flow (E. H. B. F.) within the normal range for this method.[5]

In six of the patients, establishment of a splenorenal shunt resulted in a reduction of the E. H. B. F. In one there was little change, and in two there was a slightly increased E. H. B. F. One of the latter (T. G.) has had recurrent bleeding since her operation.

COMMENT

Linton [6] and Snell [7] state that at least 50% of patients with portal hypertension will be dead within 18 months of their first hematemesis. Reduction of portal pressure and hepatic blood flow may allow a period of prolonged survival in such patients.

Production of a splenorenal shunt seems to result in reduction of liver blood flow. If portal hypertension is a consequence of increased resistence to flow through the portal venous system within the cirrhotic liver, then reduction in flow should result in a portal pressure decrease. This reduction in hepatic flow, however, is much greater than portal pressure measurements at the time of operation would suggest. Comparative measurements of liver flow may be a better index of the effect of a splenorenal shunt on hepatic dynamics. One patient (L. L.), who had an actual increase in portal pressure after completion of a splenorenal shunt, showed a sizeable reduction in hepatic blood flow by our measurements. At the time of a second operation portal pressure measurements were found to be considerably lower. Simultaneous blood flow determination was identical to the prior postoperative figures.

The two patients with greatest reduction in hepatic circulation both died in the postoperative period.

One (S. F.) did well for one week after operation; then she became comatose and died. Blood ammonia levels were elevated to toxic levels. This suggests that too large a shunt may precipitate liver coma.‡ Two patients in Bradley's series suffered from mental deterioration after shunting. It is to be noted that these two had the greatest reduction in hepatic blood flow in his series.

The second patient who died was found to have a thrombosed, nonpatent shunt at autopsy. Whether or not this occurred prior to measurement of the postoperative E. H. B. F. could not be determined.

SUMMARY

Production of a splenorenal shunt in cirrhotics usually results in reduction of estimated hepatic blood flow.

These changes seem to have little relation to changes in portal pressure measured at the time of surgery.

Dr. Robert R. Linton and Dr. Chester M. Jones cooperated in making these studies.

‡ References 8 and 9.

ADDENDUM

REPORT OF CASES

S. F. (MGH No. 768214), a 73-year-old woman, weighed 145 lb. (65.8 kg.). In good health until a sudden bout of hematemesis two years prior to admission. X-rays at that time revealed the presence of esophageal varices. She was treated with six blood transfusions and then discharged home. Her alcoholic intake had consisted of a daily bottle of beer. She remained in good health until three weeks prior to admission, when she had her second bout of hematemesis; she was hospitalized and given 2000 cc. of blood intravenously and then transferred to the Massachusetts General Hospital. At the time of admission her total protein was 5.9 gm. per 100 cc. A/G ratio was 3.3/2.6; prothrombin content was normal; sulfobromophthalein test revealed 31% retention. At operation, splenectomy and end-to-side splenorenal shunt were done, and she had an uneventful postoperative course. Liver biopsy revealed "advanced portal cirrhosis, fibrocongestive splenomegaly, and old infarct of spleen." Postoperative blood chemistry tests revealed a total protein of 5.3 gm. per 100 cc. and A/G ratio of 2.6/2.7; prothrombin content was 73%; sulfobromophthalein test, 37% retention; cephalin flocculation was 2+ in 24 hours, 3+ in 48 hours. She had an excellent postoperative course for one week, then became comatose and died in what was believed to be hepatic coma.

C. R. (MGH No. 793534), a 42-year-old man weighing 133 lb. (60.3 kg.), was admitted to the hospital because of weakness, edema, and a history of alcoholism. On physical examination he was found to have hepatomegaly, cardiomegaly, and ascites, as well as a 4+ guaiac stool. X-rays revealed large esophageal varices. He was treated with transfusions, plasma, high-vitamin-high-protein diet, and dextrose given intravenously. Because of continued gastrointestinal bleeding, a transthoracic ligation of his esophageal varices was done, from which he convalesced uneventfully. He was then discharged home, to return for shunt surgery after a period of convalescence. He failed to keep his return appointment and was then admitted to the emergency ward for a recurrence of massive bleeding. At the time of this admission the total protein was 5.6 gm. per 100 cc., with an A/G ratio of 2.5/3.1. The prothrombin content was 63%; cephalin flocculation was 2+ in 24 hours, 3+ in 48 hours. A splenectomy and end-to-side splenorenal shunt were done. Pathological report revealed "portal cirrhosis and fibrocongestive splenomegaly." His postoperative course was stormy and complicated, and he died one week after operation. At autopsy the shunt was found to be thrombosed and nonpatent.

L. L. (MGH No. 798643), a 16-year-old girl, had a bout of abdominal pain and hematemesis one month prior to admission. She was taken to a hospital where a diagnosis of acute appendicitis was made, but laparotomy revealed a normal appendix and a small bowel full of dark blood. The liver was found to be nodular. She was transferred to the Massachusetts General Hospital, where, because of continued bleeding, a thoracotomy with transesophageal suturing of esophageal varices was carried out. Blood studies revealed a total protein of 7.3 gm. per 100 cc., with an A/G ratio of 3.7/3.6. Prothrombin content was 57%; cephalin flocculation was 3+ in 24 hours and 4+ in 48 hours. Sulfobromophthalein test showed 38% retention. Her postoperative course was uneventful, and she was discharged to a nursing home for a period of two months and then readmitted, at which time a splenectomy and end-to-side splenorenal shunt were done. Pathological report was "postnecrotic portal cirrhosis and fibrocongestive splenomegaly." Her postoperative course was uneventful, and she did well for a period of six months. She then had another bout of hematemesis and was rehospitalized. It was thought that her previous splenorenal shunt might have thrombosed, and the patient underwent a reexploratory operation. However, the shunt was found to be patent. Liver biopsy at the time of this operation was reported as "portal cirrhosis." Although she convalesced uneventfully from her operation, the liver disease progressed, and she died three months later.

C. G. (MGH No. 801492), a 50-year-old man weighing 143 lb. (64.9 kg.), complained of three years' transient ascites, weight loss and melena and two bouts of hematemesis—one requiring seven transfusions. On physical examination he was found to have hepatomegaly and splenomegaly. X-rays showed esophageal varices. Total protein was 7.8 gm., with an A/G ratio of 4.2/3.6. Prothrombin content was 73%; sulfobromophthalein test showed 17% retention. A splenectomy and end-to-side splenorenal shunt were done. Pathological report revealed "portal cirrhosis and fibrocongestive splenomegaly." The patient's postoperative course has been uneventful, and he has remained in good health without further hematemesis for more than one year.

C. S. (MGH No. 26410), a 59-year-old man weighing 129 lb. (58.9), had a history of a daily intake of 1 qt. (0.94 liter) of wine for 40 years. Physical examination revealed splenomegaly and hepatomegaly. X-rays revealed the presence of esophageal varices. Hematological studies confirmed a diagnosis of Cooley's erythroblastic anemia. At operation a splenectomy was done, and, because of elevated portal pressure as well as the clinical findings and history, an end-to-side splenorenal shunt was also done. Preoperative chemistry tests revealed a total protein of 7.2 gm., with an A/G ratio of 2.3/4.9. Sulfobromophthalein test showed 12% retention of the dye. He has had an uneventful postoperative course.

R. B. (MGH No. 795946), a 67-year-old woman weighing 154 lb. (69.9 kg.), complained of anorexia, weight loss, and postprandial fullness of one year's duration. During this period she had several bouts of hematemesis and melena. X-ray studies revealed esophageal varices and splenomegaly. Blood chemistry tests revealed a total protein of 8.1 gm., with an A/G ratio of 4.2/3.9. Prothrombin content was 63%, and sulfobromophthalein test showed 18% retention. A splenectomy and end-to-side splenorenal shunt were done. The pathological report was "portal cirrhosis." The patient had an eventful postoperative course and was living and well, without evidence of recurrence, more than a year after operation.

T. G. (MGH No. 807835), a 45-year-old woman weighing 134 lb. (60.8 kg.), had had three bouts of massive hematemesis, 11, 6, and 3 months prior to admission. X-ray examination revealed esophageal varices. There was no history of excessive alcoholic intake. Blood chemistry tests revealed a total protein of 6.2 gm., with an A/G ratio of 3.9/2.3, and a prothrombin content of 57%; sulfobromophthalein test showed 30% retention of the dye, and a cephalin flocculation test was 2+ after 24 hours and 2+ after 48 hours. A splenectomy and end-to-side splenorenal shunt were done. Pathological report revealed "portal cirrhosis and fibrocongestive splenomegaly." She did very well postoperatively for two months, but then she had another bout of hematemesis. She had continued to do moderately well since then on conservative medical management.

H. B. (MGH No. 827526), a 49-year-old man, weighing 132 lb. (59.9 kg.), had a history of being jaundiced 20 years prior to admission and a history of diabetes. He had a consistently high alcoholic intake. He apparently had had one bout of massive gastrointestinal bleeding due to esophageal varices. At the time of admission, x-rays confirmed the presence of esophageal varices and chemistry tests revealed a total protein of 17.3 gm. per 100 cc., with an A/G ratio of 2.5/4.8. Sulfobromophthalein retention was 23%, and the prothrombin content was 50%. Because of evidence of severe liver disease, he was transferred to a nursing home for a month and then readmitted, at which time a splenectomy and end-to-side splenorenal shunt were done. Pathological report revealed "portal cirrhosis, congestive splenomegaly with siderotic nodules of lymph nodes." His postoperative course was complicated by hemorrhage and wound infection. However, he has had no further recurrence of hematemesis, although his liver disease has not permitted his complete rehabilitation.

L. W. (MGH No. 803698), a 45-year-old man, had a three-year history of anorexia, jaundice, and malaise. In the six months prior to admission he had had two bouts of melena. X-ray examination revealed esophageal varices. Blood chemistry tests at the time of admission revealed a total protein of 7 gm. per 100 cc., with an A/G ratio of 3.2/3.8. Prothrombin content was 63% of normal, and there was 32% retention of sulfobromophthalein. Cephalin flocculation was 3+ in 24 hours and 4+ in 48 hours. At operation a splenectomy and end-to-side splenorenal shunt were done. Pathological report revealed "postnecrotic cirrhosis and fibrocongestive splenomegaly." His postoperative course was very smooth, and he has remained well without further bleeding for more than a year.

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Treatment of Hirschsprung's Disease with Entire Colon Involved in Aganglionic Defect

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In our experience with 150 patients having Hirschsprung's disease, 2 patients have been encountered in whom the entire colon was deficient in ganglion cells. Diagnosis and treatment of this condition required variations from that followed in the usual cases of Hirschsprung's disease where the aganglionic segment is limited to the rectum and rectosigmoid.

The two patients with deficient Auerbach's plexus ganglion cells throughout the colon presented so bizarre a clinical picture in comparison with that typically encountered in congenital megacolon, that they posed a considerable diagnostic problem. Both of these infants were admitted to the hospital because of abdominal distention and vomiting. Roentgenograms with barium revealed the colon to be essentially normal in size, the abdominal distention being due to small bowel dilatation. There was some attempt to empty the colon; however, a portion of the barium was retained in the colon and was still there in one patient several months after an ileostomy was performed. The diagnosis was established at the time of laparotomy by biopsy of the right colon and was confirmed by rectal biopsy. Initial operative therapy in both infants was ileostomy, after which they did well. In spite of the infants' satisfactory progress, the question was raised by the families whether or not the ileostomy would be a permanent arrangement.

In considering the method of eliminating the ileostomy, experience with one other child having megacolon suggested the definitive treatment finally performed for these infants. This child did well with an ileoproctostomy after doing poorly after a conventional subtotal colectomy.

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REPORT OF CASES

CASE 1.—B. J. (BFH #16971) had a sigmoid colostomy at 8 days of age because of clinical symptoms suggesting obstruction in the distal colon. When he was 2½ years old, the sigmoid colostomy was closed. A fecal fistula developed; abdominal distention became pronounced, and constipation was severe. A diagnosis of Hirschsprung's disease was made. At 4½ years of age, the terminal ileum was anastomosed to the rectosigmoid colon just above the pelvic floor, followed by subtotal colectomy. The child never did well after this operation. Fistulas developed from the site of the anastomosis, producing deep abdominal abscesses. Intermittent intestinal obstruction was severe enough at times to necessitate passage of a long intestinal tube for decompression. Solution of the problem was not entirely clear, since no mechanical obstruction could be demonstrated. As a desperate measure an ileostomy was performed when the child was 6 years old.

After ileostomy, the child made a remarkable recovery in general health. In considering the question of how to eliminate the ileostomy, the only possibility available was to resect the remaining aganglionic pelvic colon and to perform anastomosis between ileum and the distal cuff of rectum. At age 7 years, the operation was performed in a manner similar to resections of this region in patients with congenital megacolon. A loop ileostomy remained proximal to the anastomosis until healing was complete. The spur was then crushed and the ileostomy closed extraperitoneally. Postoperatively the patient developed chronic fever associated with trou-

blesome diarrhea and distention. The ileostomy was reopened, and he promptly regained his health. When he was 8 years old, the ileostomy was closed again, this time with resection of the loop and the performance of an end-to-end anastomosis. The boy continued to do well after this operation. He had occasional bouts with abdominal distention relieved by insertion of a rectal tube and release of considerable quantity of gas. This treatment was less frequently used as he grew older, and has not been necessary in four years.

The patient has been followed five years since the last surgical procedure. For four years he has been essentially without symptoms. His weight gain has been normal. He has no abdominal distention and passes two to three semiformed stools daily. There has been no incontinence. Recent roentgenograms of the ileum by way of barium enemas showed filling of multiple loops of bowel occupying the lower two-third of the abdomen (Fig. 1). The mucosal pattern was that usually noted in the ileum, but the intestinal loops appeared slightly wider than normally seen. The distal 6 to 8 in. was quite dilated and without observable peristalsis. This segment appears to have taken on the function of a reservoir.

It was on the basis of the satisfactory course of this patient after ileoproctostomy that the same operation was performed in two recent cases in which the aganglionic defect of Hirschsprung's disease extended throughout the colon. The initial procedure was a loop ileostomy when the infants were a few days old. By making the ileostomy proximal to the physiologically obstructed colon, normal function of the alimentary tract was obtained. After several months of normal development, the ileostomy was resected and a total colectomy performed. The rectum was dissected free of surrounding structures down to the sphincter mechanism. The rectosigmoid colon was divided after being crushed and stapled with a von Petz clamp. The rectum was then inverted out through the anus. The distal end of the ileum was pulled through the inverted rectum and anastomosed to the anus. A two-layer anastomosis was used: the first, interrupted fine nonabsorbable surgical (silk) sutures to the muscular coats; the second, interrupted absorbable (gut) sutures to the mucosa. When the anastomosis was finished, it retracted 2.5 cm. above the anal sphincter.

Fig. 1 (Case 1).—Barium enema performed in May, 1954, five years after ileoproctostomy.

Fig. 2 (Case 2).—Photograph several months after ileoproctostomy shows normal nutrition and development.

Both babies did well postoperatively, taking a full formula within a few days. Initially, there were as many as 12 loose stools a day, and the care of the perineum was a time-consuming nursing problem. However, in a few weeks the number of stools decreased to four loose movements a day. The gain in weight and growth compare favorably with standard charts of infant development.

Case 2.—J. W. (BFH #18545), a 5-day-old boy, was admitted because of bile-stained vomiting and progressive distention from birth. On examination he was normally developed. The abdomen was distended and had increased peristaltic sounds with high-pitched tinkles. There was no meconium in the rectum. Roentgenograms of the abdomen showed generalized distention of small bowel loops. Barium

Fig. 3 (Case 3).—Photograph one year after ileoproctostomy shows normal growth.

enema demonstrated no particular abnormality of the colon.

A laparotomy was performed Aug. 6, 1952. There were many dilated loops of small bowel extending down to the terminal ileum which was small in diameter. No ganglion cells were found in biopsies of the right colon; a right colectomy was performed and a loop ileostomy made. Tissue removed at the time of internal anal sphincterotomy also showed no ganglion cells in the myenteric plexus.

The infant had an uneventful postoperative recovery. Growth was satisfactory over the next few months. Care of the ileostomy was troublesome to the parents. Four months later, a left colectomy was done. Two weeks after this second operation the ileostomy was resected and the terminal ileum was pulled through the anus and anastomosed to the rectal cuff 2.5 cm. from the mucocutaneous margin. Histologic study of the entire colon failed to reveal any ganglion cells in the myenteric plexus. The frequent loose stools with perineal excoriation gradually subsided during the first few weeks after operation.

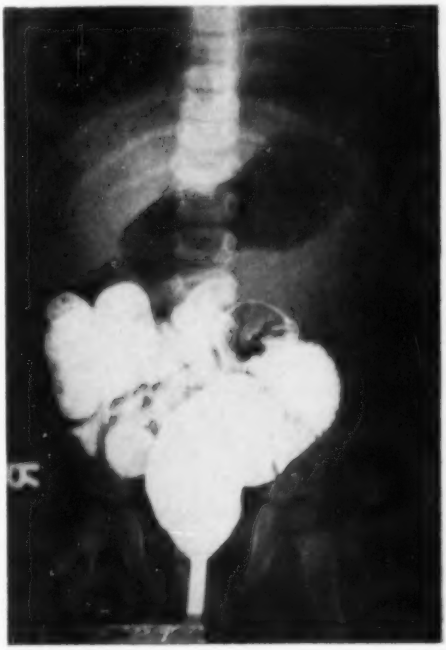
The weight gain and growth have been steady (Fig. 2). The postoperative follow-up is now two years. There has been no incontinence.

Case 3.—D. McG. (BFH #19246), was admitted the day after his birth because of vomiting and abdominal distention. Colonic irrigation gave meconium returns. On examination there was generalized abdominal distention, with tympany and decreased peristaltic sounds. X-ray study of the colon by barium enema showed the right colon to be dilated and filled with meconium and gas. Barium passed to this area and was evacuated. An intravenous pyelogram failed to demonstrate any abnormality.

Laparotomy revealed a perforation of the colon, thought to be in the sigmoid region. A loop ileostomy was performed. Tissue removed from the internal anal sphincter and rectum contained no ganglion cells. The infant did well postoperatively. The ileostomy functioned normally and distention subsided. The ileostomy was troublesome for the parents to care for. However, the weight gain and development were normal. When 3 months old, he contracted bronchopneumonia which responded to therapy. At 5 months of age an episode of pyelitis due to Aerobacter aerogenes was controlled with antibiotic therapy.

When the child was 7 months old, the ileostomy was resected and a total colectomy performed. The terminal ileum was anastomosed to the rectum 2.5 cm. from the mucocutaneous margin utilizing the pull-through technique. No ganglion cells were found in the myenteric plexus throughout the colon. The postoperative course was satisfactory. As high

Fig. 4 (Case 4).—Barium enema performed in May, 1954, one year after ileoproctostomy.



as 11 stools a day in the early postoperative period were well tolerated with minimum excoriation of the perineal skin. At 1 year of age the child had torsion of the left testis.

This boy has now been followed one year since colectomy. He is in excellent health (Fig. 3). He is having only three stools a day. There is no abdominal distention. Roentgenogram of the lower bowel by barium enema showed the loops of ileum filling the lower half of the abdomen (Fig. 4). The bowel segments visualized appeared normal in caliber, perhaps slightly wider than usually seen. The barium was poorly evacuated.

COMMENT

From the experience with these three unusual cases of Hirschsprung's disease, it would seem that the colon may be resected and the ileum anastomosed to what is essentially the anal canal, provided the ileum is normal. We firmly believe that this is not a procedure to be used in patients with ulcerative colitis. We have attempted ileoproctostomy on one patient having this disease, with near disaster. Within six weeks, emergency ileostomy was necessary because of pyrexia and profuse bloody diarrhea. Ulcerative colitis is probably a generalized disease with occult changes in the small intestine and, therefore, the procedure cannot be safely used to rid the ulcerative colitis patient of his ileostomy.

Colectomy with anastomosis of the ileum to the sigmoid or rectosigmoid colon have been reported in the literature as operative treatment of Hirschsprung's disease. Satisfactory results were obtained, but obstructive complications similar to those in Case 1 were recorded.*

Anastomosis of the ileum directly to the anus in Hirschsprung's disease has not been reported previously. This operation removes the functional obstruction caused by the long segment of aganglionic bowel. It is of value primarily in those cases in which the entire colon has a defective myenteric plexus. It is advisable to determine by biopsy that the terminal ileum has normal ganglion cells before the anastomosis is made, since it is conceivable that the aganglionic process may extend into the ileum.

SUMMARY

Three cases of Hirschsprung's disease treated by total colectomy and ileoproctostomy are reported. On the basis of satisfactory results from this form of surgical treatment, the operation is recommended in cases of Hirschsprung's disease where the aganglionic defect in the myenteric plexus extends throughout the colon.

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Anal Ileostomy

Experiences with a New Three-Stage Procedure

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Total colectomy until recently has meant a permanent ileostomy. However, several authors * have recommended preservation of the terminal rectum, which is then fulgurated of polyps or stripped of the mucosa in the area accessible to a sigmoidoscope, thus permitting ileosigmoidostomy. Such a procedure is intended to save the terminal rectum and yet prevent the subsequent development of malignant disease therein.

In 1947, Ravitch and Sabiston [6] described a one-stage surgical procedure in dogs for accomplishing an anal ileostomy, which they thought might be feasible in man with some modification. Procedures of this type have since been applied to patients with either ulcerative colitis or polypoid adenomatosis by Ravitch and co-workers,† Wangensteen and Toom,‡ Best,§ and Devine and Webb.[14] A summary of the 29 cases of these investigators as tabulated by Best in 1952 [13] shows satisfactory results in about half of the patients with ulcerative colitis and in less than half of the patients with adenomatosis or malignant growth.

Because of this relatively small experience with anal ileostomy, the severe postoperative complications, and the high percentage of unsatisfactory results, reports of additional observations appear desirable. The four cases in the present report illustrate certain of the complications following a one-stage anal ileostomy and a promising method for overcoming these difficulties.

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‡ References 10 and 11.

§ References 12 and 13.

REPORT OF CASES

CASE I.—L. W., a 28-year-old single Negro woman, was admitted to the Homer G. Phillips Hospital with a six-month history of intermittent diarrhea, bloody stools, weakness, malaise, right lower quadrant pain, and a 25 lb. (11.3 kg.) weight loss. Past history was not significant, and physical examination revealed only a slightly distended abdomen with left lower quadrant tenderness. On sigmoidoscopic examination a hemorrhagic friable mucosa with numerous ulcers was noted. A barium enema showed evidence of ulcerative colitis. Stool examinations were positive for blood and mucus and negative for amebae. Other laboratory tests were not significant. The patient received whole blood transfusions, succinylsulfathiazole (Sulfasuxidine), penicillin, and vitamins. She improved and was discharged two months after admission.

She was readmitted four months later with complaints similar to those on initial admission, as well as swelling and pain in her legs and hands. She was again discharged, improved, two months after second entry, after therapy similar to that administered during her initial hospitalization.

She reentered the hospital after four additional months because of persistent diarrhea and melena and again received whole blood and antibiotic therapy. About six weeks after this third admission the terminal ileum and all of the colon to within 2 in. (5 cm.) of the mucocutaneous junction were resected. The mucosa of the remaining rectum was curetted, and a pull-through anal ileostomy was performed. The postoperative course was stormy; the diarrhea could not be controlled, and the distal 1 in. (2.5 cm.) of ileum sloughed. There was also mild but persistent bleeding from the terminal 1 in. of large intestine. The patient developed several episodes of electrolyte imbalance, corrected by parenteral replacement.

The diarrhea and bleeding subsided gradually, and she was discharged from the hospital 13 weeks

after operation. She was subsequently hospitalized once with an acute gastritis, but she is at present working and on no special diet. There is no leakage or perianal skin irritation. The patient is on no special diet, has regained the weight loss, and has six to eight controlled, voluntary liquid or semisoft bowel movements per 24 hours.

Case 2.—H. D., a 22-year-old white woman, was admitted to The Jewish Hospital with a history of marked diarrhea during the five days preceding entry, as well as 17 lb. (7.7 kg.) weight loss during the previous six months. Physical examination revealed only a flat abdomen, with tenderness and rebound pain in the left lower quadrant. Proctoscopy showed a markedly spastic rectum with markedly inflamed mucosa which was ulcerated and bloody. Laboratory findings were not significant except for stool examinations, which revealed pus and blood, and no ova or parasites were found. She remained in the hospital for almost two months and responded to dietary and symptomatic therapy. On discharge she felt somewhat improved.

She reentered about 10 months later, during which time she had lost 6 lb. (2.7 kg.). For the previous six months she had passed four or five bloody, formed stools per day. She complained of anorexia and intermittent cramps. She remained in the hospital for four months and again responded fairly well to symptomatic therapy. However, she returned one month later because she was having ten to fifteen bloody bowel movements a day and was suffering from abdominal cramps, tenesmus, nausea, and anorexia. She had lost an additional 7 lb. (3.2 kg.). One month after this admission an ileostomy was performed, which was followed by an improvement in general health. However, the temperature remained moderately elevated, and a discharge of pus by rectum continued. A barium enema showed definite evidence of progression of the ulcerative colitis. During this period of hospitalization she showed progressive symptomatic improvement, and she was discharged five months after the operation.

During the following eight years this patient was admitted to the hospital seven times for various complaints related to the ulcerative colitis. She continued to have cramping pains, diarrhea, and intermittent attacks of fever. On the sixth admission there was evidence of fistula formation and pseudopolyposis, and, following entry for the seventh time, total colectomy and anal ileostomy were performed. The colon was markedly thickened and inflamed, and there was a stricture at the hepatic flexure. Numerous polyps were found throughout the large bowel, and an abscess was noted between the leaves of mesentery of the gastrocolic omentum. A perforation was found retroperitoneally behind the descending colon. She subsequently developed a retroperitoneal abscess on the left side, which was incised and drained. Afterward she was up and around but was having numerous bowel movements uncontrolled by diet or medication. Eleven months after surgery she continued to have numerous bowel movements, but there was no leakage. During the next three months she improved progressively and gained weight, although she experienced tenesmus and diarrhea during the later afternoon.

She was discharged during the latter period but was readmitted four months later with a four-day history of pain in the right flank and fever. Examination revealed an abscess in the right retroperitoneal space, which was drained, and antibiotic therapy was instituted. Improvement was progressive, and she was discharged one month later.

She has since shown a progressive gain in weight and has 8 to 12 bowel movements a day but no leakage. She is working eight hours a day as a stenographer.

Case 3.—H. L., a 33-year-old white man, first noted blood in his stools nine year ago, while in the U. S. Army. At that time he had about five soft stools per day. His condition was diagnosed as ulcerative colitis, and he was discharged one year later. He did fairly well for about one year, when after abandoning his bland diet he again noted blood and mucus in his stools. He was hospitalized two years after discharge, at which time proctoscopic examination revealed multiple ulcerations and sessile polyps of the rectum. Patient was subsequently admitted to the local V. A. hospital at frequent intervals for routine check-ups, during which period he continued to have five to eight soft to loose stools per day, frequently associated with mucus and pus. But he maintained his weight and experienced no chills or fever.

At one routine check-up about three years before this paper was written, proctoscopic examination revealed four small sessile polyps in the terminal 4 in. (10 cm.) of the large bowel. No ulcers were seen, but stool examinations revealed blood. A barium contrast enema also revealed a polyp higher up in the descending colon. Because of the malignant potentialities of these polyps and the long history of ulcerative colitis, it was decided to employ surgical intervention. Accordingly, total colectomy and anal ileostomy were performed. The patient tolerated the procedure well, but his appetite was poor postoperatively. For the first three weeks there was no control of bowel movements. He subsequently developed acute abdominal pain and vomiting. Insertion of a rectal tube gave relief, but he again became distended and began to vomit, and his condition became progressively worse. An emergency laparotomy was performed, and about 6 liters of fecal-smelling thin purulent fluid was removed

from the peritoneal cavity. No definite site of abscess or perforation was found. The lower quadrants and pericolic recesses were drained. He did well postoperatively, treatment consisting of continuous Wangensteen suction, whole blood transfusions, parenteral fluids, and antibiotics. On the third postoperative day there was audible peristalsis, and within the next two months his appetite improved, and there was a marked decrease in the amount of purulent drainage.

About a month later the abdominal drainage sites were completely healed, but he continued to have abdominal cramps and distention and appeared to have a chronic partial small bowel obstruction despite dilatation of the anal opening several times a week. Two months later he developed an abscess caused by gas-forming organisms in the right hip which was believed to have resulted from undermining along the fascial planes from the region of the internal sphincter. A stab wound permanent ileostomy was placed in the right lower quadant and drainage of the perianal region instituted. The pain in the hip disappeared along with the abdominal cramps, and he began to gain weight.

He continued to improve, but subsequent examination showed the ileostomy stoma to be small, and it had to be revised. He was discharged seven months after surgery but returned four months later with an indurated, hot, erythematous area around the ileostomy stoma; the ileum was practically flush with the skin. A catheter was placed in the ileostomy opening until the skin irritation had subsided. He was then placed under general anaesthesia; the ileum was brought to the outside and transplanted to another position, away from the abdominal scars. Subsequent course was uneventful, and he gained weight. He was discharged one month after the ileostomy revision.

Five months later he again entered, and an abdominal perineal excision of the terminal ileum and rectal stump was performed. Except for a temporary period of impotence, treated with 17-methyltestosterone (Metandren Linguets), he has remained well for the past year.

CASE 4.—B. R., a 23-year-old married woman, was admitted to the Homer G. Phillips Hospital complaining of frequent loose stools containing mucus and blood. Bleeding had been profuse for a three-week period prior to hospitalization, and she had noted progressive weight loss and weakness. Past history and physical examination revealed nothing of significance, but sigmoidoscopic examination showed a friable rectal mucosa with numerous flat grayish ulcerations. A barium enema showed roentgenological evidence of ulcerative colitis. Laboratory examinations were not contributory, except for stools which showed mucus and blood but no ova or parasites. Despite whole blood transfusions and dietary measures she became progressively worse and ran a febrile course.

Six weeks after entry, after preparation of the large bowel for surgery, about 6 in. (15.2 cm.) of terminal ileum and the colon to the distal sigmoid were resected through a long left paramedian incision, and a right lower quadrant stab wound ileostomy was performed. The proximal end of the defunctioned distal colon and rectum was brought out through the lower angle of the left paramedian wound. The patient improved rapidly, gained weight, and was discharged from the hospital on the 45th postoperative day.

She was readmitted 10 weeks later. At this time sigmoidoscopic examination revealed numerous pseudopolyps extending to the mucocutaneous junction. The left paramedian scar was excised, and the abdominal cavity was reentered. The remaining sigmoid and rectum to about 5 cm. from the mucocutaneous junction was excised, and the mucosa of the remaining large bowel was curetted. The ileostomy was taken down, and an anal ileostomy was performed. A loop of ileum was selected and transected. A double-barelled ileostomy was formed; the proximal limb was brought out through the previous right lower quadrant stab wound, and the distal limb was brought out through the left paramedian incision at the same level as the stab wound. On the fifth postoperative day the anal ileostomy was revised. Postoperative course was uneventful, and the patient was discharged on the 42d postoperative day.

She was again admitted 10 days later and was able to retain 100 cc. of water instilled into the distal limb of the ileum for about four and one-half hours. The two ileal openings were circumcised and joined by an end-to-end ileoileostomy. The patient was subsequently discharged, having 8 to 10 controlled liquid stools every 24 hours.

At present, 12 months after the last surgical procedure, she is working and on no special diet. She has three to four semisolid stools during the day and three to four similar bowel movements at night. There is no anal incontinence.

COMMENT

The search for an adequate technique for accomplishing an anal ileostomy is motivated by a desire to avoid the disagreeable features of a permanent ileostomy. However, experiences to date with anal ileostomies have revealed several important problems. In the immediate postoperative period stool urgency and loose movements are difficult to control even though continence may be quite satisfactory. Perineal excoriations may develop

and persist for several weeks after operation, but with final healing of the suture line these disappear or lessen to a degree so that the patient is comfortable. With a competent sphincter a complete although intermittent obstruction frequently occurs and patients suffer agonizing cramps during the period of dilatation of the sphincter. Fortunately, in most instances, this is only temporary and is followed later by compensation.

Healing of the anal ileostomy suture line presents a major problem. Breakdown and leakage of the suture line results in the formation of perianal fistulas or abscesses which usually require permanent ileostomy. Two failures by Ravitch || were due to necrosis of the distal ileum with the development of large abscesses. In several cases reported to date, as in Case 3 reported herein, abdominal perineal resection with permanent ileostomy had to be performed because of such severe breakdown of the suture line.

The problem of preserving sphincteric control deserves special mention. In this regard the investigations of Gaston ¶ indicate that the rectum and its intact sensory nerve supply are an integral part of the normal control of the sphincteric mechanism; however, this nerve supply does not appear to be impaired by fulguration or stripping of the mucosa. Even though the sphincter and its motor supply are preserved and it can be contracted voluntarily, unless a sufficient portion of the rectum with its sensory nerve supply is left intact following surgical excision, sphincteric continence will not result. Gaston's investigations further indicate that at some point between 1 and 7 cm. there is a level below which the rectum cannot be resected without serious impairment of sphincteric control. Utilizing a different technique, Goligher and Hughes [18] have made the important observation that balloon distention which produces the rectal type of sensation evokes a contraction of the external anal sphincter that appears to be partly voluntary and partly reflex.

In reviewing the subject of anorectal function and disease, Turell, Krakauer, and Maynard [19] conclude that in the performance of sphincter-saving operations, if sensory incontinence is to be avoided, it is mandatory to preserve undamaged not only the sphincter muscles, but also at least a 6 cm. and preferably an 8 cm. or more anorectal stump with its parasympathetic nerve supply. The rectal wall with its parasympathetic innervation may be preserved by resecting the mucosa of the rectum and anastomosing to it the terminal ileum. The technique of anal ileostomy as applied by Ravitch and colleagues,# Wangensteen and Toom,* Best,† and Devine and Webb [14] is based on this principle. However, judging from the results in successful cases reported by these investigators, as well as those in the present series, a rectal segment of about 5 cm. (approximately 2 in.) appears to be sufficient to regain sphincteric control.

Further, Gaston noted that in one of his cases with anal ileostomy, although the patient could retain ileal contents by sphincteric contraction, he could not do so for longer than one hour at a time, so that he had to go to stool hourly throughout the day and night. In the present group of successful cases, as well as those of others referred to above, the ileum was eventually able to take over the function of a reservoir. Bowel movements continue to be more numerous than in normal persons, but eventual equilibrium is attained at a level of about 6 to 12 bowel movements per 24-hour period.

From the standpoint of final outcome, 3 of the 4 cases reported herein may be considered successes, thus raising the number of satisfactory results reported in recent years from 17 to 20 as opposed to 13 unsatisfactory results. However, Cases 1 and 2 of the present report illustrate many of the postoperative complications incident to anal ileostomy. It was because of these difficulties that the technique reported was used.

|| Ravitch, M. M.: Personal communication to the author.

¶ References 15-17.

References 7-9 and Ravitch, M. M.: Personal communication to the author.

* References 10 and 11.

† References 12 and 13.

These complications appear to have been avoided to a large extent in Case 4. The procedure used was based on the principle of diverting the fecal stream from the anal ileostomy suture line until complete healing was assured. In the first stage, resection of about 6 in. (15 cm.) of terminal ileum and all of the colon to the distal sigmoid was carried out and a right lower quadrant stab wound ileostomy was performed; the proximal end of the defunctioned sigmoid was brought out through the distal angle of the left paramedian wound. In the second stage, the rectosigmoid was excised to within 2 in. (5 cm.) of the mucocutaneous junction and the distal remnant was curetted. At the same time, the ileostomy was taken down and a pull-through anal ileostomy was performed. A double-barreled ileostomy was then constructed. In the third stage, after the function of the distal ileum was determined by noting that the patient was able to retain 100 cc. of water instilled therein for over four hours, an ileoileostomy was performed.

The foregoing plan would be especially valuable for patients who are in relatively poor condition. For those who can withstand more intensive surgery the following plan will be utilized: Stage 1 will consist of colectomy, pull-through anal ileostomy, and diversionary proximal ileostomy; revision of the anal ileostomy will be carried out five to seven days later, when indicated. Stage 2, ileoileostomy, will be performed four to eight weeks later, as determined by the ability of the patient to retain 100 cc. of water instilled into the distal limb of the ileum for at least four hours.

Anal ileostomy for the diseases mentioned has been abandoned by many surgeons because even the "successful" cases are often characterized by stormy postoperative courses. This has been particularly true in patients with ulcerative colitis, less so in those with familial polyposis. The operation carried out in the fourth case gives promise of greater success, although further experience is required.

SUMMARY

Four cases are presented in which anal ileostomy with total colectomy for ulcerative colitis was done. In the first three, severe postoperative complications occurred, necessitating, in one, a final permanent abdominal ileostomy; the other two ileostomies eventually healed and developed good function and remain satisfactory.

In the fourth case the procedure was done in stages, the essential feature of which is a temporary ileostomy at the time of anal-ileal anastomosis, thus permitting the latter to heal with exclusion of fecal contact. There were no complications in this case, and the clinical result was excellent, with restoration and maintenance of body weight and excellent sphincter control; this patient has now been observed for more than 12 months.

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Reinforcement of Fresh Autogenous Venografts to Aorta

Experimental Study with Polyvinyl Formalinized (Ivalon) Sponge

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Successful replacement of segments of major arteries or of the aorta with preserved arterial homografts has given new impetus to the development of vascular surgery. In addition to extending the indications for resection of segments of arteries or of the aorta, successes with homografts have stimulated efforts to develop other methods for replacing the resected vessels.

Utilization of a segment of fresh autogenous vein as a graft to replace a resected segment of artery has certain advantages over the use of preserved arterial homografts. First, the use of a fresh autogenous venograft obviates the necessity of an arterial bank, which under many circumstances is difficult or impractical to maintain. Second, the fresh autogenous venograft is reported to "take," remain viable, and grow in its new position without being replaced by the recipient's tissues. These more biologically acceptable autografts have thus been considered less likely to undergo degenerative changes and might therefore be expected to give better long-term results than arterial homografts which, in the final analysis, are foreign bodies that must be replaced with the recipient's own tissue.

Use of portions of veins as grafts to replace resected segments of large arteries or of aorta, however, has certain practical and theoretic inadequacies. One of the most important of these drawbacks is the danger of dilation and formation of an aneurysm in the relatively thin-walled, inelastic segment of vein which, when implanted into the aorta or a large artery, must carry blood under arterial pressures. Several investigators* have reported a high incidence (50% or more) of dilation of such venografts applied to the aorta. Generally good results in so far as absence of thrombosis, continued viability, and freedom from degenerative changes in these venografts have prompted numerous attempts to reinforce the walls of the venograft in the hope of overcoming its weakness and preventing dilation. Fascia, pericardium, cutis grafts,[5] and fibrous tubes produced by plastic surgery † have been used as reinforcing materials but with only poor to mediocre results. Dilation, constriction, infection, and increased tendency toward thrombosis were the usual complications in such reinforced venografts. Vargas and Deterling[6] have recently reported favorably on the use of nylon netting for reinforcement of venografts to the aorta.

The aim of these various methods of reinforcement is to produce a pliable tough wall

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which will act as an accessory adventitia to the relatively thin-walled venograft. The strength of the new adventitia is derived partly from the material used and partly from fibrous tissue which replaces or invades it. In no instance has elastic or smooth muscle tissue been induced to develop in this newly formed adventitia.

The unique properties of polyvinyl formalinized (Ivalon) sponge which enable it to become thoroughly impregnated with tough yet pliable and vascularized fibrous tissue, thus forming a "living reinforcement," prompted us to consider this material as a possible reinforcing agent around fresh autogenous venografts implanted into the aorta. After successful preliminary pilot investigations by one of us (J. H. G.), the following study was carried out to determine the feasibility of applying fresh autogenous venografts reinforced with polyvinyl formalinized sponge to the aorta.

MATERIALS AND METHOD

Operative Procedure (Fig. 1).—Adult female mongrel dogs weighing about 15 kg. were used. The anesthetic agent, pentobarbital sodium (Nembutal), was administered intravenously, and the right external jugular vein was exposed through a longitudinal cervical incision. A segment of this vein, 7 cm. in length, was isolated and removed. It was placed in sterile physiologic saline solution which was at room temperature. The cervical incision was closed. A long midline abdominal incision was then made; the abdominal viscera were retracted, and the posterior peritoneum was incised to expose the abdominal portion of the aorta. The branches arising from the distal 8 cm. of this part of the aorta (including the inferior mesenteric and usually three or four pairs of lumbar arteries) were ligated and divided. Blalock clamps were then applied to the aorta, the proximal clamp being placed about 2 cm. distal to the left renal artery and the distal clamp just above the trifurcation. A 4-cm. segment of aorta between these clamps was then removed, and the segment of fresh autogenous jugular vein was used as a free vascular graft to bridge the defect in the aorta. Anastomoses were

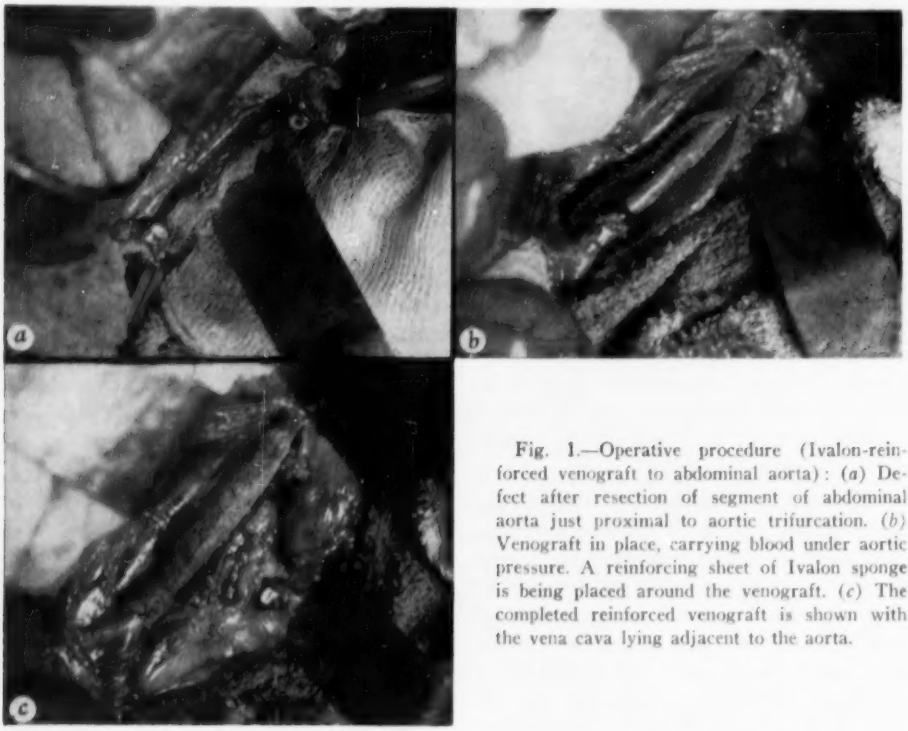

Fig. 1.—Operative procedure (Ivalon-reinforced venograft to abdominal aorta): (*a*) Defect after resection of segment of abdominal aorta just proximal to aortic trifurcation. (*b*) Venograft in place, carrying blood under aortic pressure. A reinforcing sheet of Ivalon sponge is being placed around the venograft. (*c*) The completed reinforced venograft is shown with the vena cava lying adjacent to the aorta.

accomplished by a continuous simple coaptation suture of No. 00000 oiled (nonabsorbable) surgical silk. Clamps were then removed from the aorta, allowing blood to flow through the venograft. When necessary, hemostasis was achieved by pressure or an occasional interrupted suture. When the field was entirely dry, the venograft was inspected and was demonstrated to be patent, with good flow, good pulsation, and normal contour.

If the venograft was not to be reinforced, the posterior peritoneum was closed at this point, and the abdominal incision was closed in layers. If reinforcement was to be carried out, the reinforcing material was applied in the following manner: a rectangular sheet of the reinforcing material measuring 4 by 8 cm. was prepared and was wrapped around the graft as a sleeve, its edges being approximated anteriorly by a running suture of No. 5-0 silk. Care was taken to make this reinforcing sleeve fit snugly over the venograft but not compress it. Each end of the reinforcing sleeve extended 1 cm. onto normal aorta and was anchored to the aorta with interrupted sutures. The reinforcing sleeve was placed in position and sutured without the use of clamps on the aorta, the venograft thus carrying blood under aortic pressure while its reinforcement was being applied. The posterior peritoneum was then closed over the graft, and the abdominal incision was closed in layers.

Postoperative Management.—Anticoagulants were not administered. The dogs were inspected daily for one week, but special postoperative care was not given. If the flow of blood through the graft appeared to be impaired (manifested by the absence of femoral pulsations or by paralysis of the hind legs), an aortogram was done. If impairment of the flow of blood was not evident, aortograms were done routinely after 4 to 12 weeks and again 5 to 9 months after the operation.

Types of Reinforcement.—Since this study was carried out to investigate the possibility of using polyvinyl formalinized surgical sponge ‡ as the reinforcing material, most dogs (23) received grafts that were wrapped with this material. The sponge used was 2 mm. thick, with dimensions as previously described. The width of the sheet of sponge was determined by wrapping it around a solid rod with a diameter the same as that of the dog's abdominal aorta under normal conditions (that is, about 1 cm.). It was then cut so that its edges would just meet around the rod, giving a snug fit without tension on the closing suture line. The sponge, of course, was soft and pliable, having been soaked in isotonic sodium chloride solution.

‡ The surgical Ivalon sponge used in this study was furnished by the manufacturer, Ivano Inc., Chicago, and may be obtained on order from Clay-Adams, Inc., New York.

About every third dog operated on was used as a control, the venograft either not being reinforced (in two dogs) or being wrapped with other reinforcing material. In three dogs fresh autogenous fascia was used, this being obtained from the anterior sheath of the rectus abdominis muscle at the time the abdomen was opened. The fascia was applied in the same manner as the sponge, the fibers running longitudinally over the graft, not circumferentially. In three other dogs both sponge and fascia were used, the fascial sheet being applied directly to the venograft, while the sponge was used as the second, or outer, covering. Venografts in three dogs were reinforced with nylon netting obtained from Dr. R. A. Deterling, who had used similar material in previously reported studies.[8]

Methods of Postoperative Evaluation.—Since the terminal portion of the abdominal aorta was the site of the grafts, the clinical condition of the dogs could not be utilized as a criterion of the patency of the graft. In three dogs, in which thrombosis occurred within a few days after operation, paralysis or weakness of the hind legs had developed in association with the absence of femoral pulses. However, all other dogs had palpable femoral pulses and used their legs normally, even though several had completely occluded grafts, the blood reaching the legs via collateral channels. Therefore, aortography was utilized to evaluate the grafts in living dogs. In addition, in all dogs to be killed, the intact grafts were examined at surgical exploration while they were still carrying arterial pressure, in order that the texture, pulsations, and external contour of the graft might be evaluated under living conditions. When the dogs were killed, the site of the graft was studied grossly; its elasticity, thickness of the walls, contour, diameter, length, and color were evaluated, and the presence or absence of thrombi was noted. Histologic sections were made of every graft, and studies were carried out with hematoxylin and eosin stain and the Verhoeff method for elastic tissue.

TABLE 1.—*Status of Dogs With Venografts to Aorta*

Venograft, Type of Reinforcement	Number of Dogs			
	Died After Operation	Killed After Aortography 8 to 250 Days After Operation	Alive for Long-Term Studies	Total
Ivalon	1	14	8	23
None	0	1	1	2
Fascia	0	2	1	3
Fascia and Ivalon	0	2	1	3
Nylon netting	0	3	0	3
Total	1	22	11	34

Eight dogs with Ivalon-reinforced venografts and three control dogs were permitted to live, to be examined later in long-term studies.

RESULTS

Clinical Sequelae.—A total of 34 dogs received autogenous venografts to the abdominal aorta (Table 1). One dog died, a mortality rate of 3% for this operation. In this dog, which had an Ivalon-reinforced venograft, evisceration through the abdominal incision occurred 72 hours postoperatively. The graft was in excellent condition. Weakness or paralysis of the hindlegs developed in three other dogs (one with an Ivalon-reinforced graft, one with a fascia-reinforced graft, and one with a graft reinforced with both Ivalon and fascia) during the first week after operation. Aortography showed evidence of complete occlusion of the grafts, and these animals were killed on the eighth or ninth postoperative day. The remaining 30 dogs were well and healthy seven to nine months after operation, without clinically recognizable sequelae from the operative procedures.

Evaluation by Aortography.—Results were classified first as either good or poor (Table 2). To be considered a good result the graft had to be patent and have a regular contour with a lumen essentially the size of the lumen of the aorta above and below the graft (Fig. 2). Constriction, dilatation, irregularity of contour, mural thrombi, or narrowing at anastomoses was sufficient to cause the result to be classified as poor, even though impaired flow of blood through the graft may not have been evident (Fig. 3). Of course any evidence of occlusion at the site of the graft caused the result to be classified as poor.

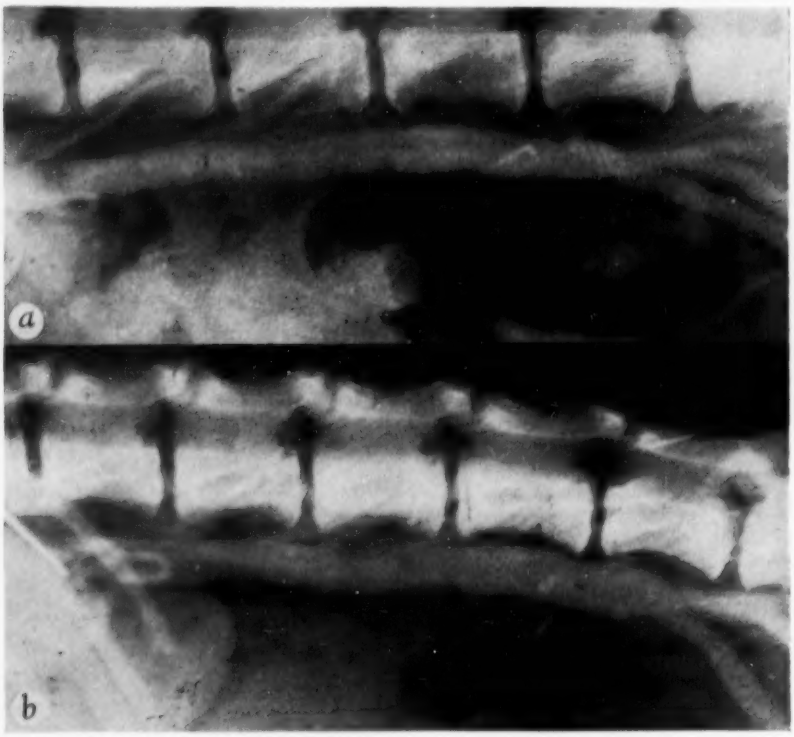

Fig. 2.—Aortogram of an Ivalon-reinforced venograft to aorta showing good results: (*a*) View of aortogram three months after implantation of an Ivalon-wrapped autogenous venograft 7 cm. in length in abdominal aorta. (*b*) Aortogram seven months after operation.

By these rigid criteria, good results were obtained in 17 grafts (52%), evaluated seven to nine months after the operation. Fifteen of the good results were obtained in the 22 dogs (68%) which had venografts reinforced with Ivalon sponge. Good results were obtained in only 2 of 11 dogs (18%) which had other types of venografts; 1 of these had an unreinforced venograft, the other had a venograft reinforced with fascia.

Further analysis (Table 2) of the poor results revealed that in 6 of the 16 dogs with poor results complications could be attributed to technical errors during surgical implantation of the graft and were probably not a failing of the graft itself. Such technical errors included constriction at anastomoses in four, use of too long a graft in one, and use of too short a graft in another. Correction for these errors in technique gave final percentages for poor results for the grafts themselves as follows: 14% for Ivalon-reinforced grafts, 50% for unreinforced venografts, and 67% each for venografts reinforced with fascia, with nylon netting, and with both fascia and Ivalon.

The 10 poor results attributed to the grafts themselves consisted of the following complications (Table 3): Thrombosis occurred in three dogs; two had Ivalon-wrapped venografts, and one had a venograft wrapped with both fascia and Ivalon.

Stricture of the graft occurred in three dogs. In these cases the lumen of the graft was narrowed to pinpoint size and was occluded with fibrous tissue. It is possible, of course, that this represents contraction of an organized thrombus. The constriction, however, was not present in the first postoperative aortograms but developed progressively within three to six months after operation. We are inclined to interpret these instances of constriction as representing excessive fibrosis around the graft, with resultant narrowing owing to cicatricial contraction. Another possible factor which we have not been able to evaluate could be excessive tension on too short a venograft, resulting in constriction due to longitudinal tension. Of these three cases of constriction of the graft, one occurred in a fascia-wrapped venograft and two in nylon-netting-wrapped venografts.

Dilatation occurred in four dogs. In one the venograft was reinforced with both fascia and Ivalon sponge; the longitudinal suture line closing the sleeve of Ivalon was insecure,

TABLE 2.—*Results of Venografts to Aorta*

Venograft, Type of Reinforcement	Dogs Studied	Good Results		Poor Results					
				Technical Error		Graft Itself		Total	
		No.	%	No.	%	No.	%	No.	%
Ivalon	22	15	68	4	18	3	14	7	32
None	2	1	50	0	0	1	50	1	50
Fascia	3	1	33	0	0	2	67	2	67
Fascia and Ivalon	3	0	0	1	33	2	67	3	100
Nylon netting	3	0	0	1	33	2	67	3	100
Total	33	17	52	6	18	10	30	16	48

TABLE 3.—*Poor Results Attributable to Venografts*

Venograft, Type of Reinforcement	Complication				
	Thrombosis	Stricture	Dilatation	Tortuosity	Total
Ivalon	2	..	1	..	3
None	..	..	1	..	1
Fascia	..	1	1	1	3
Fascia and Ivalon	1	..	..	..	1
Nylon netting	..	2	..	..	2
Total	3	3	3	1	10

allowing separation of the reinforcing layer, with consequent dilatation. This, of course, was a technical error, and the poor result could not be attributed to the graft itself. In the other three instances dilatation was evidently attributable to inability of the reinforced vein to withstand aortic pressure. One instance of dilatation occurred in the unwrapped venografts, one in the fascia-wrapped venografts, and one in the Ivalon-wrapped venografts.

Finally, tortuosity of the venograft occurred in one dog. In this case, the venograft was reinforced with fascia. Tortuosity was not present in the early postoperative aortogram but developed after three months and progressed thereafter. This we consider to be secondary to the scarring and fibrous contraction resulting from the fascial reinforcement.

Hemorrhage from a graft, sepsis, formation of abscess, or recognizable peripheral emboli were not noted.

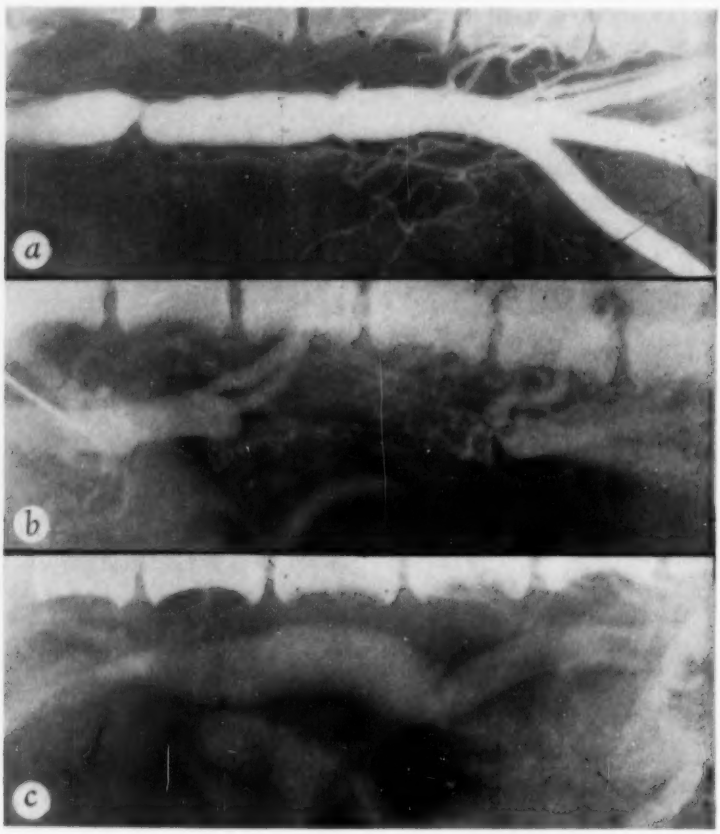

Fig. 3.—Three aortograms showing poor results obtained from venografts to aorta: (*a*) Venograft wrapped with Ivalon sponge, six months after operation; note constriction at anastomotic suture lines. (*b*) Venograft wrapped with nylon netting, six weeks after operation (note complete occlusion of graft, which was found at necropsy to be thrombosed). (*c*) An unreinforced venograft to aorta, four months after operation (note dilation of graft).

Pathologic Evaluation.—Gross Findings: Except at sites of intimal damage (such as that produced in the aorta by the occlusive clamps), marked changes in contour, or changes in diameter of the lumen (such as that caused by narrowed anastomoses), little tendency toward the development of thrombi

in these venografts was evident. In all grafts which were patent the inner surface was grossly shiny and smooth (Fig. 4). We could not recognize any relationship between intimal changes and different methods of reinforcement of the wall of the vein. Mild inflammatory reaction, attributable to the surgical procedure itself, was recognizable around unreinforced venografts. A similar degree of inflammatory reaction and ultimate fibrosis occurred around the Ivalon-wrapped

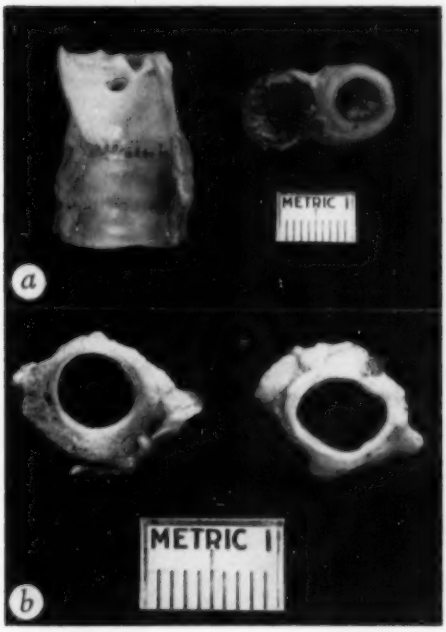

Fig. 4.—Gross appearance of Ivalon-reinforced venografts to aorta, six months after implantation: (*a*) Left, inner surface of graft at anastomosis; aorta above and graft below; right, cross section of graft and adjacent vena cava (note relative thickness of wall of venograft and the vena cava). (*b*) Cross section of Ivalon-reinforced venograft on left (compare with normal aorta on right). Note relative thickness of wall of venograft and normal aorta.

venografts. The inflammatory reaction around fascia-wrapped venografts, however, was somewhat increased, and that around nylon-netting-wrapped venografts was even more marked. Within three months the wall of the venograft itself, unreinforced or reinforced and regardless of the method, was thickened about fourfold, so that its thickness was essentially that of the normal aortic wall. The reinforcing layer in all instances was firmly adherent to the venograft, adding to its over-all thickness (Fig. 4). A sheet of fibrous tissue also developed outside the reinforcing sleeve, this fibrous coat attaching firmly to the adventitia of the aorta above and below the venograft. The unreinforced venografts were soft and pliable and pulsated well, although not with the same expansile pulsation as the normal aorta. The reinforced venografts were all less pliable and firmer than the normal aorta, depending on the degree of fibrosis present. All grafts which were patent pulsated palpably. Elasticity of the venograft was never as great as that of the aorta, but differences in elasticity could not be attributed to the different methods of reinforcement.

Microscopic Changes: Evidently the intima of the venograft disintegrated soon after implantation, as it was not recognizable in grafts 72 hours after operation (Fig. 5*a*). However, six weeks after operation a layer of fibrous tissue several cells thick lined the inner surface of the venograft (Fig. 5*b*). This new intima extended across the anastomosis and fused with the intima of the aorta. After the graft had been in place for four to nine months, this intimal layer had condensed considerably and contained more collagen (Fig. 5*c*). At times the cellular elements of the newly formed intima resembled smooth muscle cells, but at no time was elastic tissue identified in it. Much of the thickness of the healed venograft wall consisted of this newly formed intima.

In the first week after implantation the media of the venograft appeared acellular, as though infarcted. Its elastic fibers were easily identifiable, and the distance between the individual fibers was normal. After three months, the media of the original venograft was still recognizable, but it was considerably thinner than when it was implanted. Elastic tissue was still readily identifiable. The distance between these elastic fibers,

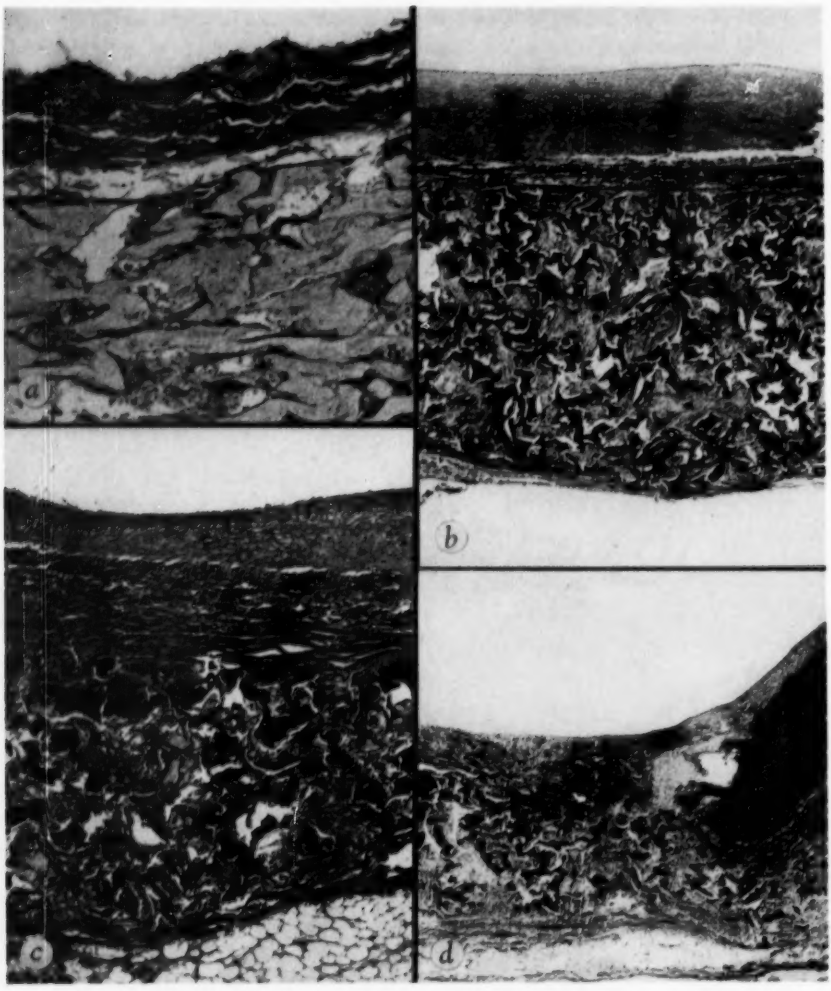

Fig. 5.—(*a*) Microscopic appearance of Ivalon-reinforced venografts to aorta 72 hours after implantation into aorta (hematoxylin and eosin; × 110). Note absence of intima, the elastic fibers of the media forming the inner surface of the graft. Note also the polymorphonuclear leucocytes in the interstices of Ivalon sponge. (*b*) An Ivalon-reinforced venograft six weeks after implantation (Verhoeff's stain for elastic tissue; × 30). Note newly formed, thick intima, thin media with a few elastic fibers, and abundant vascular fibrous tissue in interstices of Ivalon sponge. (*c*) An Ivalon-reinforced venograft eight months after implantation (Verhoeff's stain for elastic tissue; × 60). Note thickened intima and condensed Ivalon sponge impregnated with fibrous tissue. Only a few elastic fibers of the media of the original vein remain. (*d*) Region of anastomosis of Ivalon-reinforced venograft to aorta eight months after implantation (Verhoeff's stain for elastic tissue; × 40). Note the smooth fusion of the intima of the normal aorta with the newly formed fibrous intima of the venograft. The fibrous tissue in the Ivalon sponge around the venograft continues onto the aorta as a firmly attached outer layer. The elastic fibers of the media of the aorta, however, end abruptly and have no attachment or continuity with the media of the venograft. Venograft to left; aorta to right.

however, now varied. In some instances the distance was normal, suggesting that either preexisting tissue between the elastic fibers had not been removed, or, if it had been removed, that it had been replaced by a comparable amount of new tissue. In other areas the elastic fibers lay close together, suggesting that the other elements of the media had disintegrated and disappeared. Although the media of the venograft and that of the aorta lay in apposition at the anastomoses, this layer showed little tendency to fuse (Fig. 5*d*).

Within three months after implantation of the venografts an adventitia had developed consisting of vascular connective tissue which mingled with the fragments of adventitia of the original vein. This outer layer of fibrous tissue was fused with the adventitia of the aorta beyond the venograft. When Ivalon sponge was used for reinforcement, the interstices of this material became infiltrated with neutrophilic granulocytes within 72 hours after implantation. Vascular fibrous tissue gradually infiltrated these interstices, this fibrous tissue being continuous with the adventitia of the venograft and with an outer layer of fibrous tissue which developed external to the sheet of Ivalon. Similar fibrosis occurred in and around fascial reinforcements. Elastic fibers in the transplanted fascia were identifiable as long as six months after implantation. Calcium salts were recognized in the Ivalon sponge about three months after implantation, and the amount had increased somewhat after eight months.

Little change was noted in the histologic characteristics of the venografts between four and nine months after implantation. The connective tissue became somewhat more dense, with less vascularity and greater deposition of collagen, and the Ivalon material seemed to decrease somewhat in thickness, but otherwise the venografts seemed stable and unchanged during this interval.

We were unable to demonstrate that the elements of the original venograft in any instance continued to grow normally or retain their cellular integrity. Some of the elastic fibers of the media could be demonstrated nine months after implantation and the reinforcing material remained intact, but all other elements of the venograft appeared to have developed from the recipient.

SUMMARY AND CONCLUSIONS

Segments of fresh autogenous external jugular vein of dogs were used as free grafts to replace resected segments of the abdominal portion of the aorta. Such venografts, when reinforced with a sheet of polyvinyl formalinized (Ivalon) sponge wrapped around the vein at the time of implantation, have functioned satisfactorily and remained remarkably free from tendencies to thrombose, dilate, or constrict during a nine-month follow-up period. Poor results attributable to the graft itself were found in only 3 (14%) of 22 Ivalon-wrapped venografts to the aorta. On the other hand, similar fresh autogenous venografts which were not reinforced or which were reinforced with fascia, with nylon netting, or with both fascia and Ivalon sponge were less satisfactory, more than 60% (7 of 11) of such venografts giving poor results.

Histologic evidence was not present to show that these fresh autogenous venografts to the aorta continued to grow or to maintain their cellular integrity. On the contrary, it appeared that all constituents of the healed venograft wall, with the exception of a few elastic fibers in the media and the reinforcing exogenous material, had developed from the recipient's own tissues in a manner similar to that occurring in arterial homografts.

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Sacrococcygeal Cysts and Sinuses

Use of Elastic Pressure Dressing to Obliterate Dead Space and Facilitate Primary Healing

GORDON M. CARVER Jr., M.D., Durham, N. C.

Obliteration of dead space created by block excision of a sacrococcygeal cyst or sinus offers a challenge to the skill of the surgeon attempting a primary closure. Many ingenious techniques have been devised to close this dead space,* as it is the key to primary healing.

The problem of wound healing in the sacrococcygeal region is complex, and infection frequently nullifies the results of the most meticulously performed surgical procedure. The presence of numerous sweat glands and hair follicles makes preliminary sterilization of the skin almost impossible, and late contamination of the wound is a hazard because of excess perspiration and the proximity of the anus. Tissue trauma produced in mobilizing flaps tends to promote the exudation of fluid, and the sacral fascia offers a poor absorptive surface, allowing the accumulation of fluid and blood, which is a perfect culture medium for any bacteria that gain entrance into the wound.

The operative technique to be presented was specifically designed to counteract these unfavorable factors by the use of an elastic traction mechanics waste pressure dressing, which aids the prevention of tissue fluid accumulation by obliterating dead space and relieving tension on the suture line without cutting or necrosing the skin.

OPERATIVE TECHNIQUE

Preoperatively patients receive one of the broad-spectrum oral antibiotics to minimize infection and warm sitz baths or compresses to promote drainage from the sinus tracts. Enemas are given preoperatively to remove all fecal material and obviate the necessity for bowel movements for three or four days postoperatively. If inadequate draining sinuses or abscess formation is present, preliminary incision and drainage are performed on an outpatient basis. This is done under local anesthesia, and operation is deferred until all signs of acute inflammation have subsided.

The patient is placed in the jackknife position after spinal anesthesia, and the operative field is exposed by placing adhesive strips from the skin of the buttocks to the operating table. Skin is prepared with hexachlorophene (pHisoHex), ether, and 10% tincture of benzolkonium (Zephiran), with the anus draped out of the operative field. A double elliptical incision is made about the cyst and sinuses after injection of all sinuses with an aqueous solution of methylene blue. The skin to be sacrificed is then grasped with Kocher forceps, and upward traction is applied as the block of tissue containing the cyst and sinus is carefully dissected from the presacral fascia with a scapel.

Subcutaneous tissue flaps are mobilized for a distance of approximately 8 to 10 cm., with the plane of dissection on the presacral and pregluteal muscle fascia. Hemostasis is accomplished by electrocoagulation, and the wound is frequently irrigated with a warm saline solution containing 1,000,000 units of aqueous penicillin per liter. Elastic skin tapes are released, allowing the subcutaneous gluteal flaps to come together over the sacrum.

Four to six No. 20 stainless steel wire sutures are placed through the flaps approximately 6 cm. from the skin edge and through the entire width of the presacral fascia. Small plastic or metal buttons are threaded on the wire sutures, and a row of interrupted chromic 0 (gut) absorbable surgical sutures is placed to approximate the leading inferior edges of the subcutaneous flaps together and to the presacral fascia. The skin is closed with a continuous Halsted subcuticular No. 26 Stainless Steel wire suture, and the stay sutures are adjusted to relieve the tension on the inner (gut) layer of sutures. Eight rubber bands capable of exerting approximately 10 to 15 lb. (4.5 to 6.8 kg.) of pressure

From the Department of Surgery, Duke University School of Medicine.

* References 1 through 7.

are then tied to the stainless steel wire stay sutures, and a double handful of mechanics waste is slipped under the rubber bands (Figure).†

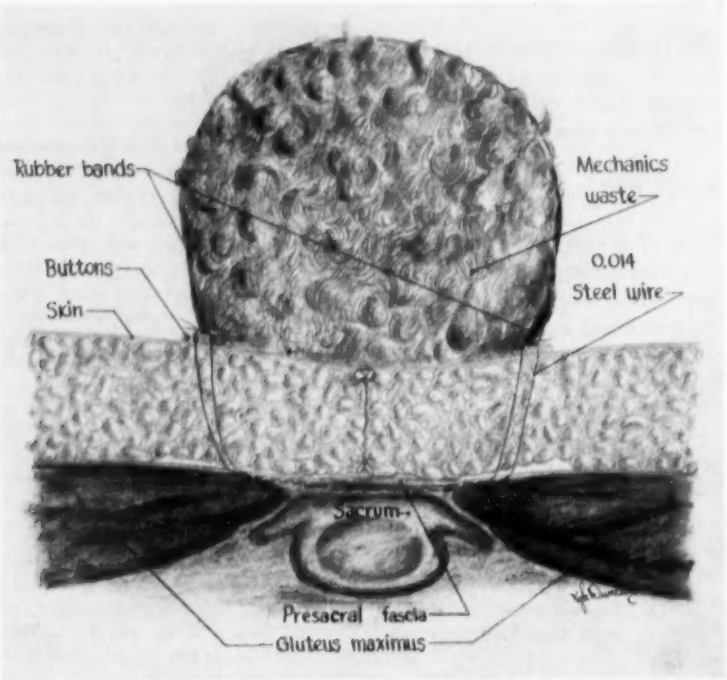
Postoperatively patients remain on their abdomen or side for the first 24 hours. Then they are allowed to walk but not permitted to sit. Antibiotic therapy is maintained, and a low-residue diet is prescribed. On the third postoperative day patients are given an enema, and care is taken not to spread the intergluteal folds or wet the pressure dressing. Female patients are instructed to urinate in the standing position. Patients experience little pain postoperatively and require analgesics infrequently. When pain is experienced, it usually means that fluid is accumulating under the mobilized flaps and that infection is developing. Infections are treated by the insertion of a Penrose drain into the wound without removing the pressure dressing.

The wire stay sutures and the Halsted subcuticular suture are removed on the eighth postoperative day. Only the small stay suture wire holes are visible on the skin. There is no cutting or necrosis of the skin from the stay suture because of the counteracting pull of the rubber bands. After removal of the sutures patients are instructed to avoid the sitting position and direct trauma to the sacral region for at least 10 days.

RESULTS

In order to determine the applicability of this technique in the treatment of simple as well as complicated sacrococcygeal cysts and sinuses, 115 consecutive patients were operated upon without regard to size of cyst, presence of lateral or perianal sinuses, recent infection, or the number of previous attempts at operative excision.

There were 10 recurrences in 115 cases, a recurrence rate of 8.6%. In most cases failure to excise the cyst or sinus tract completely, because of its association with the coccyx, lat-



Cross section through sacrococcygeal region, showing position and course of stainless steel wire sutures. Large mechanics waste dressing and rubber band countertraction obliterate dead space and prevent wires from cutting skin.

† Rubber bands used measure 3×⅛×1/16 in. (7.6×0.32×0.16 cm.) and are usually available in all hospital stockrooms.

eral or perianal sinuses, or scar tissue from previous operations, was responsible for the recurrence. There were only 4 recurrences in 88 cases in which a simple cyst or sinus was present (4.5%).

In four instances it was necessary to remove the coccyx, since a sinus tract could be followed into or through the coccygeal bone. There was one recurrence in this group of cases (25%). In four cases with perianal sinuses and in five cases with lateral sinus tracts operations were attempted, with a recurrence rate of 25% and 40%, respectively. In 14 patients who had previous operations 2 recurrences developed (14.3%).

There were nine postoperative infections, an incidence of 7.8%. Of these cases, four developed a recurrent sinus, indicating an incomplete removal of the cyst or sinus as the basis for the infection and recurrence. Primary healing was present in 105 of 115 cases at the time of removal of the sutures on the eighth postoperative day. There were no cases of skin edge pressure necrosis, and it was remarkable to note the absence of tissue edema at the operative site.

All cases were followed for a minimum of two and one-half years. The first year patients returned to the office for physical examination, and the two- and three-year follow-ups were made by letter or return visit. Ninety per cent responded to a questionnaire at the second- and third-year interval. Of the 10 cases that recurred, 8 recurred within one month, 1 at three months, and 1 at eight months after operation.

COMMENT

Four basic things are accomplished by the use of properly applied mechanical pressure to wounds:

(1) Elimination of dead space
(2) Control of oozing
(3) Limitation of venous and lymph stasis
(4) Limitation of the amount of plastic material that pours into the wound.[8]

These principles, outlined by Blair in 1924, have specific application in the sacrococcygeal area for promoting primary wound healing after the excision of a sacrococcygeal cyst or sinus. Previous methods of application of mechanical pressure over the wound ‡ have certain disadvantages. When adhesive tape is used, pressure is inefficient and excessive sweating and skin sensitivities occur. Vertical mattress retention sutures tied over mechanics waste results in necrosis of the skin.[1] The rubber band countertraction dressing exerting its force over the mechanics waste changes the angle of tension at the site of entrance of the stay sutures through the skin so that there is less tendency for them to cut. This method of compression remains constant even when the patient is ambulatory. In addition, this dressing with its stay sutures relieves the tension on the central healing site, protects the wound against trauma, aids in the prevention of extracellular fluid accumulation by obliterating dead space, and aids in the prevention of infection by absorbing the ever-present contaminated perspiration of the sacrococcygeal skin.

In the surgical management of sacrococcygeal cysts and sinuses the issue is still whether or not to employ an open or closed operative procedure. An excellent discussion of the merits and applications of the various techniques used has been published by Turell and Gladstone,[9] and a detailed discussion of the subject is beyond the purview of this communication. In general, the open technique has been preferred, primarily because of a lower incidence of recurrence. In a statistical review Palumbo and co-workers [10] found the recurrence rate after open procedures to be 12.7% in 4884 cases, compared with 21.9% in 1165 cases in which primary closure was performed. Since the introduction of antibiotic drugs, military surgeons have reported successful results in more than 90% of their cases in which primary excision and closure techniques were used.‡ A recurrence rate of 8.69% in this series of 115

‡ References 1 and 3.
§ References 1 and 4.

consecutive cases is comparable to the 12.7% recurrence rate for the open operative technique reported by Palumbo and co-workers.[10]

SUMMARY

A primary operative excision and closure technique for the treatment of pilonidal cysts and sinuses is described which utilizes a mechanics waste countertraction elastic pressure dressing to promote wound healing and prevent postoperative infection.

This technique was used in 115 consecutive cases, and the recurrence rate was 8.69%. Primary healing occurred in 92.1% of the cases, and all patients were followed a minimum of two and one-half years.

Analysis of the recurrences reveals that most of the failures occurred when the sacrococcygeal cyst was complicated by lateral, perianal, or coccygeal sinuses. In 88 cases in which a simple cyst or sinus was excised and primary closure was performed, the recurrence rate was 4.5%.

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The Common Bile Duct in the Postcholecystectomy Patient

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Thirty years ago, Graham and Cole [1] published their report on a new method of visualization of the gall bladder based on the fundamental work of Abel and Rowntree.[2] Now, for the first time, a new substance is available, Cholografin, which, when administered intravenously, will produce roentgenographic visualization of the common bile duct in the presence or absence of the gall bladder.*

Cholografin is an isotonic and colorless solution of the disodium salt of N,N′-adipylbis(3-amino-2,4,6-triiodobenzoic acid) with the empirical formula $C_{20}H_{12}O_6I_6Na_2$. When first prepared by Schering, it was called Biligrafin. It contains 64% iodine which is molecularly stable and firmly bound even after injection into the blood stream. Cholografin is promptly and rapidly excreted by the liver. Its concentration in the common and hepatic ducts is sufficient for roentgen visualization within 20 minutes after intravenous injection (Fig. 1). A functioning gall bladder also may be visualized on the x-ray film one and one-half hours later (Fig. 2). Cholografin is excreted in the feces without passing through the enterohepatic circulation. If liver function is impaired, the contrast medium is excreted through the kidneys, whereupon the renal pelvis and calices may be visualized (Fig. 3).

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* References 3 through 7.

TECHNIQUE

For 12 hours prior to the examination of each patient, intake of food and fluid is restricted. In addition, a laxative such as castor oil or compound licorice powder is given the night before the examination to be followed by a saline enema the next morning. Forty cubic centimeters of a 20% Cholografin solution is injected intravenously over a period of 10 minutes. Initial films are exposed 10 minutes later and reviewed while wet. Exposures at various time intervals are made. Multiple film studies with different degrees of rotation in both erect and recumbent positions and in the lateral decubitus position are found helpful in separating shadows due to overlying ribs or gas from portions of the common and hepatic ducts. Experience has shown that 45 to 50 kv and 400 to 800 mas is desirable for marked contrast.

If the tissues adjacent to the vein were injected inadvertently, slight transient local irritation occurred. Nausea, flushing, and vomiting occurred after injection in only 6 of 50 cases. The latter reactions seem to have been due to an injection given too rapidly. Although severe reaction was not encountered, routine tests were performed for hypersensitivity before injection of the full dose of the contrast medium.

Fig. 1.—Normal common bile duct and hepatic duct. Film taken 20 minutes after the injection of Cholografin. Contrast medium can be seen in the duodenum.

Fig. 3.—Visualization of Cholografin in pelvis and calyces of kidney in jaundiced patient.

Fig. 2.—Normal gall bladder and common and hepatic ducts. Film taken 90 minutes after the injection of Cholografin.

Fig. 4.—Film demonstrating the presence of multiple calculi in common duct.

RESULTS

Fifty postcholecystectomy patients were included in this study. Of these, 40 had symptoms initially suggesting biliary tract disease. The remaining 10 patients had no symptoms.

Of the 40 symptomatic patients, clear visualization was obtained in 36 cases, or 90% (Table). Abnormalities were noted in 17 patients: stones in the common duct in 6 patients, or 15%; a dilated cystic duct stump in 6 patients, or 15%, and biliary dyskinesia in 5 patients, or 12.5%. A normal common duct was found in 19 patients, or 47.5%.

Of the 10 asymptomatic patients, 7 examinations revealed no abnormality of the common duct. In one patient, a stone was found in the common duct. The presence of this

Data on Fifty Cases Examined

Cholografin Diagnosis	With Symptoms of Biliary Tract Disease		Without Symptoms of Biliary Tract Disease	
	No.	%	No.	%
Stone in common duct	6	15	1	10
Dilated cystic duct stump	6	15	..	..
Biliary dyskinesia	5	12.5	..	..
Normal	19	47.5	7	70
Nonvisualization	4	10	2	20
Total	40		10	

stone was suspected after it had been palpated at the time of previous cholecystectomy, at which time surgical exploration of the common duct was thought inadvisable. In the remaining two patients, the common duct failed to be adequately visualized because of the presence of overlying gas shadows and colonic content.

Attempts to verify the x-ray diagnosis in this group of cases was made by surgical exploration whenever possible. Four of the six cases with common duct stones were confirmed surgically (Fig. 4). In 19 cases, the common duct appeared normal except for varying degrees of dilatation.

COMMENT

Attempts at roentgenographic visualization of the common duct were possible in the past only in the presence of an intact and function-

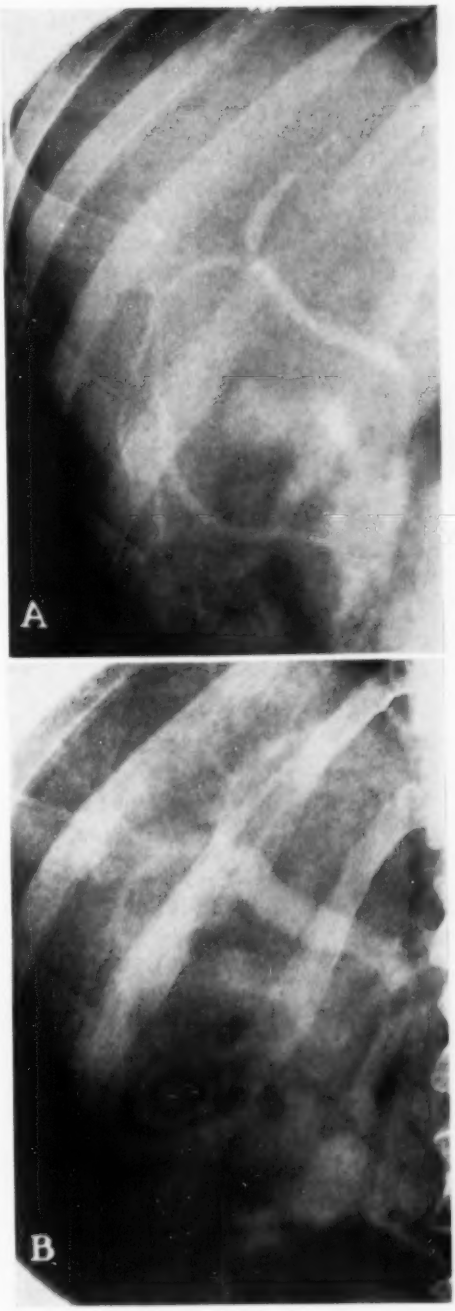

Fig. 5.—Biliary dyskenesia. *A*, common bile duct 25 minutes after injection of Cholografin; *B*, common bile duct 10 minutes later after 1/6 grain (10 mg.) of morphine sulfate was injected subcutaneously.

ing gall bladder. Cholografin now offers a means of evaluating symptoms which may originate in the common duct after removal of the gall bladder, the presence of these symptoms having been recognized as "the postcholecystectomy syndrome."

The most frequent factors contributory to this syndrome which were demonstrated in this study are biliary dyskinesia, cystic duct stump, and stones in the common duct. Biliary dyskinesia was considered to exist in the presence of spasm of the distal end of the

Fig. 6.—Cystic duct stump.

common duct, usually associated with right upper quadrant pain. When morphine sulfate was given to these patients during the examination with Cholografin, pain in the right upper quadrant was reproduced. Simultaneously, the duct became dilated, and there was a greater degree of opacification (Fig. 5). These changes were assumed to be secondary to spasm of the distal end of the common duct.

Of the six cases with oversized cystic ducts (Fig. 6), only one was reoperated. In addition to the cystic duct, microscopic examination of the tissue removed proved it to consist of a remnant of the gall bladder with changes of chronic cholecystitis. Of interest was the striking relief of symptoms of this patient following surgery. Thus, what appears to be a remnant of the cystic duct may represent, in addition, a portion of the gall bladder which, when diseased, may cause symptoms.

In spite of the selection of 40 patients who were considered to have clinical symptoms suggestive of biliary tract disease, 19 revealed no abnormality after Cholografin study. This indicates the limitation of clinical evaluation. The number of positive x-ray findings, therefore, based on symptoms pertaining to the common duct obviously will vary in accordance with the accuracy of the original clinical diagnosis.

There are limitations to the study of the common duct with Cholografin. Failure to obtain satisfactory visualization of the biliary tract can be due to liver dysfunction. In the presence of jaundice or high degree of retention of sulfabromophthalein (Bromsulphalein), the common duct was not visualized. This accounted for an inadequate study of the common duct in four cases.

Overlying gas shadows may interfere with satisfactory delineation of the common duct as was noted in two cases of this series. The intramural portion of the common duct cannot always be visualized. Hence such diseases as fibrosis of the sphincter, neoplasm, or calculi in this area may escape detection.

The size of the common duct was compared in patients before and after cholecystectomy. On the basis of this preliminary report, it appears that there is an increase in the caliber of the common duct after the removal of the gall bladder. Further studies of this phenomena are now in progress.

SUMMARY

A new intravenous medium, Cholografin, has been described which is useful for the demonstration of common duct pathology in the postcholecystectomy patient.

Good visualization of the common duct was obtained in 44 of 50 cholecystectomized patients. Various abnormalities of the common duct were demonstrated, including residual stones in the common duct, cystic duct stumps, and evidence of biliary dyskinesia. The reactions to this substance are minimal. Diagnostic failures occurred in the presence of high degree of sulfabromophthalein retention, hyperbilirubinemia, and occasional technical difficulties.

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Idiopathic Omental Torsion Simulating Appendicitis

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Infarction of the omentum as a primary disease or secondary to idiopathic torsion is considered a rare entity. Idiopathic torsion of the omentum consists of an omental twist in the absence of any recognizable etiologic factor such as a hernia, inflammatory process, or adhesions. The syndrome is clinically indistinguishable from acute appendicitis when the infarcted segment lies in the right lower abdominal quadrant. Between March 1, 1953, and March 1, 1954, 145 laparotomies were performed at the 3750th U. S. A. F. Hospital for acute appendicitis: in two cases, infarction of the omentum secondary to primary torsion was found.

REPORT OF CASES

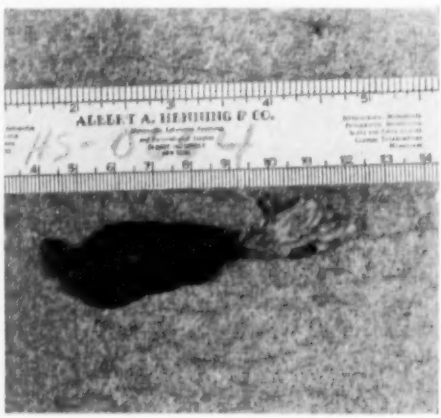
CASE 1 (Hosp. reg. no. 37 896).—G. H., a 19-year-old white airman, was admitted May 22, 1953, with a complaint of moderately severe pain in the right lower abdominal quadrant of 36 hours' duration. Although marked anorexia was present, there had been no nausea or vomiting. Two similar episodes had occurrred in the past, both of which had subsided spontaneously after two or three days. There was no change in bowel habits. Systemic review was noncontributory.

Physical examination was normal except for the abdomen, which revealed localized tenderness and rebound tenderness in the right paraumbilical region. Rectal examination revealed mild tenderness on the right, but no mass was palpable. Temperature was 100 F, and pulse rate was 80.

White blood cell count on admission was 11,600, with 70% neutrophiles; urinalysis was normal. Preoperative diagnosis was acute appendicitis.

From the Surgical Service of the 3750th U.S. A.F. Hospital, Sheppard Air Force Base, Texas.

With the patient under spinal anesthesia, exploration was performed through a transverse abdominal incision approximately one hour after admission. Serosanguinous fluid was found along with a segment of gangrenous omentum approximately 5×2.5×1.5 cm. (Fig. 1). The involved omentum was twisted at the level of infarction; this was interpreted as a volvulus. Numerous enlarged nodes were present in the mesentery. A normal-appearing appendix was removed, and the terminal 3 ft. (91.44 cm.) of ileum was inspected and found to be normal. Recovery of the patient was without incident, and he was discharged on the eighth postoperative day.



Infarcted omentum, found on exploration of a case with preoperative diagnosis of appendicitis.

Pathologic report was torsion of omentum with infarction and normal appendix.

CASE 2 (Hosp. reg. no. 42 968).—C. T., a 5-year old white girl was admitted on Feb. 23, 1954. She had been in good health until the onset of moderately severe discomfort in her lower abdomen (more marked on the right side) 24 hours before admission, which became progressively worse. The pain was constant, nonradiating, and associated with tenderness which was greatest in the right lower quadrant. She was anorexic, but there had been no nausea, vomiting, or fever. The child had slept poorly and had cried intermittently because

of her pain. Movement of any kind aggravated her pain, and she had rested with her thighs and legs flexed while lying on her back. Systemic review and past medical history were noncontributory.

Physical examination revealed a well developed and well nourished child who appeared to be in acute pain and who guarded her abdomen with her hand whenever examination was attempted. Tenderness was most marked in the right lower quadrant. Both voluntary and involuntary muscle guarding was present. Temperature was 100 F. Rectal examination did not reveal any abnormality. The remainder of the physical examination was entirely normal. A white blood cell count on admission was 14,500, with 45% neutrophiles. Urinalysis was normal. A preoperative diagnosis of acute appendicitis was made.

Approximately two hours after admission, an exploratory laparotomy was performed through a McBurney incision with the patient under general anesthesia. When the abdomen was opened, free serosanguinous fluid was encountered. A segment of gangrenous omentum, which appeared to be twisted on itself, measuring 8 cm.×4 cm.×2.5 cm., was found and resected; a normal-appearing appendix was also removed. Numerous enlarged nodes were present in the mesentery. The patient's postoperative course was complicated by mild paralytic ileus, which was successfully treated by nasogastric suction. She was discharged on the sixth postoperative day, completely asymptomatic.

The pathologic report was normal apppendix and infarction of omentum.

COMMENT

In 1946, Altemeier and Holzer [1] were able to collect a total of 64 cases of primary torsion of the omentum in a review of the literature. Our experience at this hospital would suggest that the condition is commoner.

Primary segmental infarction of the omentum without torsion is an entity which is even more uncommon. Hallstrand [2] reviewed 30 cases previously reported and added 4 additional cases in 1954.

Although many theories have been advanced to explain the etiology of infarction of the omentum with and without torsion, there is little or no evidence to support any of them.

The nonoperative treatment of omental infarction is probably attended by little danger. Because of the clinical mimicking of acute appendicitis, however, the vast majority of patients will come to laparotomy. Failure to confirm the suspected pathologic lesion at the time of surgical exploration should lead the surgeon to perform a thorough inspection of the abdominal viscera. Free serosanguinous fluid in the peritoneal cavity together with mesenteric lymphadenopathy was present in both of our cases; this has been a common finding in other cases in the surgical literature.

It is suggested that the entire omentum should be carefully examined as part of the abdominal exploration whenever laparotomy for acute appendicitis reveals an innocuous-appearing appendix. Resection of the infarcted omentum results in the prompt recovery of the patient.

SUMMARY

1. Two cases of omental infarction secondary to idiopathic torsion have been reported.

2. The clinical picture is indistinguishable from acute appendicitis.

3. Clues at surgery are the presence of serosanguinous fluid and mesenteric lymphadenopathy.

4. Careful examination of the omentum should be part of the abdominal exploration whenever the suspected pathologic lesion is not confirmed at laparotomy.

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Barbwire Tendon Suture

Clinical Experience with Thirty Flexor Tendons

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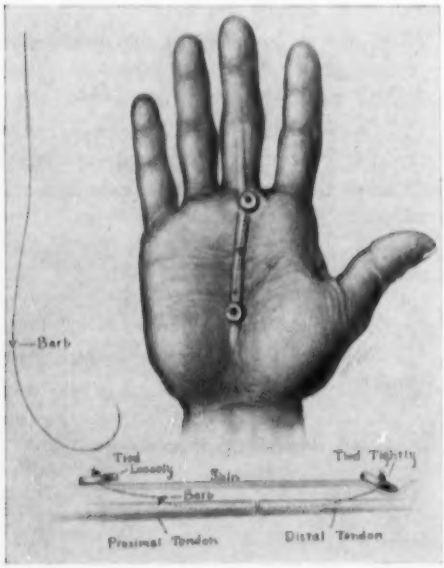
Early experiences with the barb wire tendon suture have proved it to be simple and easy to use. This suture, as previously described, seems ideal for applying the basic principles of tendon surgery. Further experiences with this technique indicate that this suture will continue to be a useful supplement for the treatment of severed flexor tendons. The purpose of this paper is to present results encountered and impressions derived from the repair of 30 consecutive freshly severed flexor tendons. The results are graded as good, fair, and poor by criteria previously expressed.*

From the Department of Surgery, University Hospital.

Fig. 1.—Details of suture (left); drawing of hand with suture in place; sagittal view (bottom).



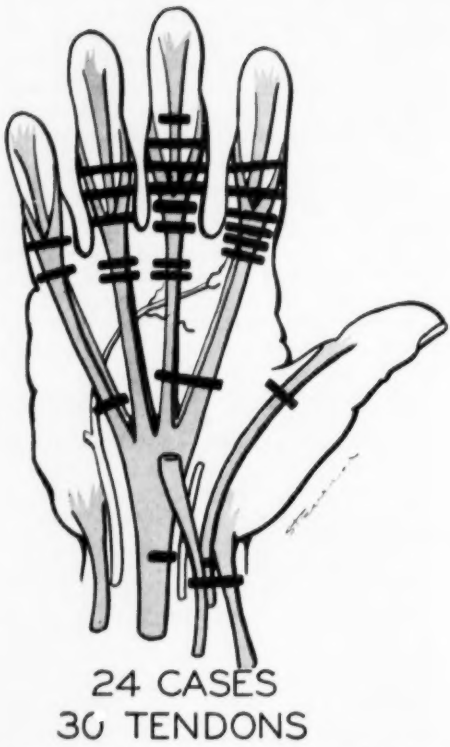


Fig. 2.—Points of severance encountered in 30 tendons (24 cases.)

TECHNIQUE

The routine preparation of the patient, as pointed out by Bunnell [3] and others, was followed. The freshly severed tendons were sutured as soon as possible in the operating room under sterile conditions. Brachial block or a general anesthetic was

* References 1 and 2.

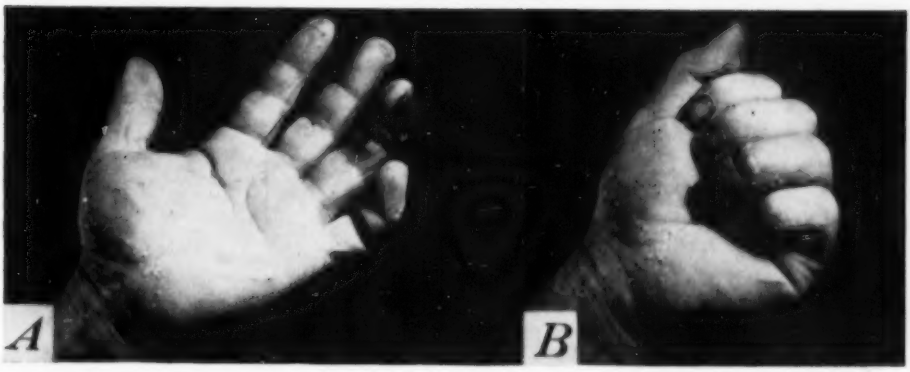


Fig. 3.—*A* (I. D.), extension—three weeks postoperatively. *B* (I. D.), flexion—three weeks postoperatively.

used. An avascular field was secured by pneumatic tourniquet. Sharp technique was used and all basic principles of hand surgery were observed. In suturing the divided tendon, the needle was first introduced into the substance of the proximal tendon segment, approximately ½ in. from the point of severance. The barb was engaged and the needle then carried into the substance of the distal segment and out through the skin (Fig. 1). The tendon ends were approximated and appropriate tension was maintained by means of a button and lead shot. The proximal portion of the suture was brought out through the skin and maintained loosely on a button. After the tendon had healed, the distal button was freed and the suture removed by pulling proximally.

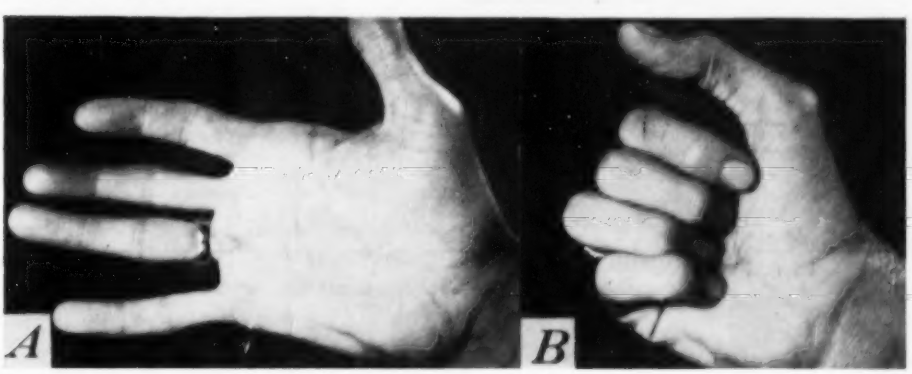
RESULTS

The results obtained are given in terms of whether or not the tendon was severed within the flexor digital sheath (Table). The actual points of severance are further shown in Figure 2. Of 30 divided tendons, 18 gave good results, 8 fair results, and 4 poor results. Of 22 tendons severed within the flexor digital sheath, only 10, or 45%, ended with good results. Therefore, it must be stressed that severed flexor tendons are still a difficult problem. Several examples of results which were classified as good are demonstrated by Figures 3, 4, 5, 6, and 7.

Results of Flexor Tendon Repair

	Good	Fair	Poor	Total
In sheath	10	8	4	22
Not in sheath	8	0	0	8
Total	18	8	4	30

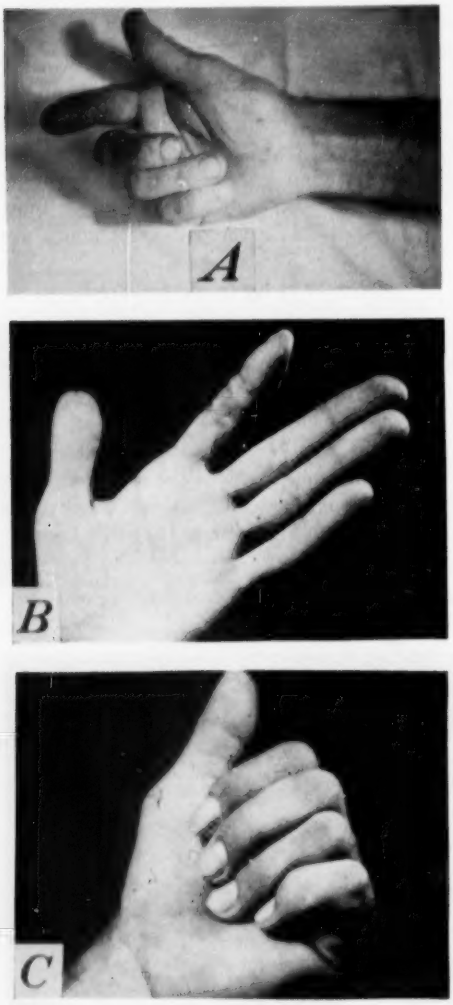
Fig. 4.—*A* (A. G.), extension three months postoperatively. *B* (A. G.), flexion three months postoperatively.



IMPRESSIONS

The barb wire tendon suture is easy to use. Certain precautions are noteworthy. The barb should be applied with care so as to substantially engage the points within the tendon substance. The barb should not be engaged too far back on the proximal tendon segment or folding of the tendon will occur. If the needle on the suture is not of the appropriate size or contour, it can be bent

Fig. 5.—*A* (J. W.), before repair. *B* (J. W.), extension four weeks postoperatively. *C* (J. W.), flexion four weeks postoperatively.



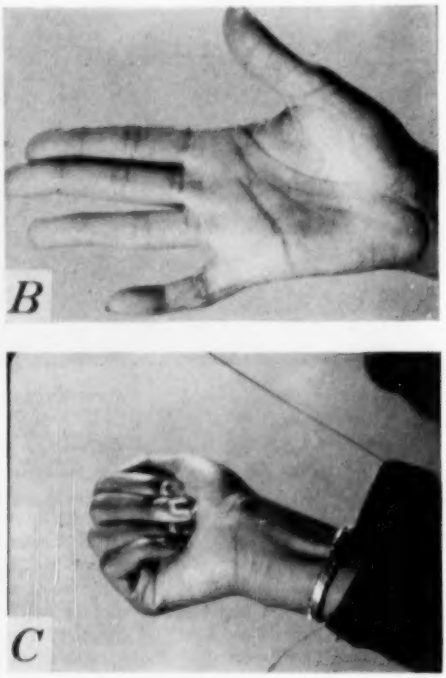


Fig. 6.—*B* (J. C.), extension four months postoperatively. *C* (J. C.), flexion four months postoperatively.

or another more desirable needle substituted. In suturing tendons within the phalanges, the distal button should be applied on the extensor surface of the fingers. Several lead shot should be used to secure the suture. The distal portion of the suture should be spaced away from joints. Early mobilization can be used, if the distal button is one joint past the point of severance.

Infection has not been a problem in this series. One hand became infected, thereby producing one of the poor results.

Tendon transplants, advancements, and grafts can be effectively done with this suture.

SUMMARY

Further experiences with the barb wire tendon suture reveal it to be a helpful adjunct in the treatment of tendon injuries.

In 30 fresh flexor tendon repairs, 18 gave good results.

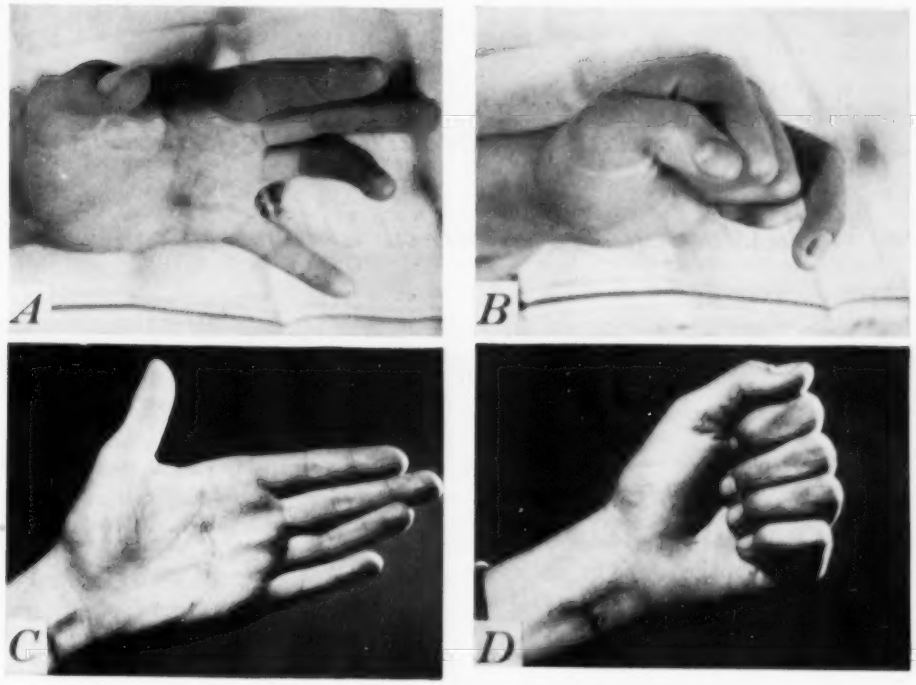


Fig. 7.—*A* (J. R.), extension before repair; *B*, flexion before repair; *C*, extension four months postoperatively; *D*, flexion four months postoperatively.

Although poor results are still being encountered, particularly with tendons severed within the flexor digital sheath, it is felt that continued use of the barb wire tendon suture will prove to be a most useful technique.

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An Anatomical Approach to the Problem of Massive Gastrointestinal Hemorrhage

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Gastrointestinal bleeding is a frequent problem and the localization of the site of hemorrhage may be a challenge. Many lines of approach have contributed information expressing the differentiation of the causes for bleeding. It was felt that the reported anatomical investigations of the problem were not extensive enough and that an additional study of an autopsy series would be enlightening. Therefore, this work was undertaken.

The importance of the gastrointestinal bleeding problem is illuminated by the observation that hemorrhage as a cause of death at autopsy at the Cook County Hospital has more than doubled since a previous review of 1938.[62]

LITERATURE

Chalmers and co-workers,[11] reporting from a similar type of charity institution, stated that in 24 of 101 necropsies, fatal gastrointestinal hemorrhage was not suspected clinically. The clinical accuracy for the diagnosis of the source of bleeding was 53%. This difficulty in ascertaining site of bleeding has been noted by others.* Crohn [18] states that 21% of his cases went undiagnosed; Schiff [73] found bleeding in 26.4% of his patients unexplained.

The inaccuracies of diagnosis must be considered with knowledge of the inherent limitations of x-ray.[21] At our own institution, in only 54% of patients with a bleeding ulcer was a roentgenological diagnosis made. The ulcer crater may be filled with clot; edema of the walls may be present; bleeding ulcers tend to be shallow and heal rapidly. For these reasons, some gastroenterologists submit their patients to study within the first few days of bleeding.† Further, gastroscopy is of limited aid except in gastric ulcer.[52] Most esophagoscopists are reluctant to examine their patients if varices are present. Therefore, the only certain means of identifying the cause of bleeding is anatomic dissection.

Sullens and colleagues,[81] reporting from this institution in 1949, gave the clinical appraisal of gastrointestinal bleeding which was

† References 32 and 72.

TABLE 1.—*Comparison of the Clinical Diagnoses in Three Hundred Five Cases of Upper Gastrointestinal Bleeding with an Autopsy Series of Six Hundred Seventy-Three Cases from the Cook County Hospital.*

Diagnosis	Sullen's et al.,[81] %	All Hemorrhage of This Series, %	Fatal Hemorrhage of This Series, %
Peptic ulcer	75.6	49.8	46.8
Portal cirrhosis of the liver	9.8	19.2	31.3
Acute gastritis	5.1	1.0	1.6
Carcinoma of the stomach	4.9	6.9	4.1
Miscellaneous causes	2.3	19.4	15.0
Diagnosis undetermined	2.3	3.7	1.2

Thesis submitted by Dr. Kane to the Graduate School of Northwestern University for a Master of Science Degree in Pathology.

From the Departments of Surgery and Pathology of the Cook County Hospital, the Northwestern University School of Medicine, and the Cook County Graduate School of Medicine.

* References 9, 11, 17, 36, and 81.

not at variance with the reports of others.‡ The marked variation between his clinical and our anatomical report is recorded in Table 1.

MATERIAL AND METHOD

The protocols of 28,400 consecutive autopsies (1929-1951, inclusive) at the Cook County Hospital were reviewed for evidence of massive hemorrhage. Hemorrhage was considered massive when the quantity of blood in the gastrointestinal tract at autopsy was greater than 500 cc., when this amount of blood, or more, was collected immediately prior to death as emesis or stool, when bleeding was sufficient to have a red cell count less than 3,000,000 or a hemoglobin less than 10 gm. per 100 cc., and no other cause for such anemia could be elicited, or when the patient went into shock owing to blood loss. Massive gastrointestinal bleeding was found in 673 cases; in 310 cases it was the primary cause of death (fatal hemorrhage); in 363 cases it was contributory or incidental to the cause of death (nonfatal hemorrhage).

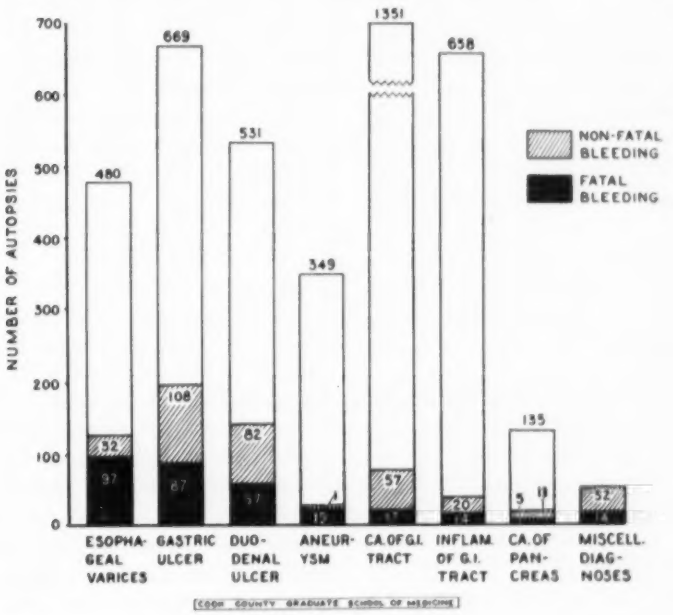
‡ References 12, 17, 36, 67, and 68.

No correlation with clinical history or laboratory data was attempted here; such a study is now in progress.

While the exact number of cases admitted to the wards with cirrhosis is not known, it is certain that this disease is unusually prevalent in this hospital population.

In compiling this information it was realized that numerous pathologists had performed and supervised these autopsies and that they frequently used different terminology and criteria. However, as all lesions were described grossly and most of them microscopically, the evaluation of the lesion could be standardized. The prosector's opinion was accepted in the evaluation of the type of bleeding, that is, whether it was the primary cause of death (fatal hemorrhage) or merely a contributory cause of death (nonfatal hemorrhage).

ANATOMIC CAUSE OF 673 CASES OF GASTRO-INTESTINAL HEMORRHAGE IN 28,400 AUTOPSIES



Graph.

OBSERVATIONS

The different anatomical diagnoses as a cause of hemorrhage are listed in the order of their incidence.

TABLE 2.—*Age, Sex, and Race of One Hundred Twenty-Nine Cases of Massively Bleeding Esophageal Varices*

	With Hemorrhage, No.	Age			Sex		Race	
		0-40	41-60	61 and Over	Male	Female	White	Negro
Fatal	97	17	61	19	75	22	79	18
Nonfatal	32	10	11	11	26	6	25	7*

* Including two Orientals.

TABLE 3.—*The Percent Incidence of Various Diseases Associated with Six Hundred Seventy-Three Cases of Massive Gastrointestinal Bleeding*

	Per Cent Incidence Usually Found	Esophageal Varix		Gastric Ulcer		Duodenal Ulcer		Carcinoma Gastrointestinal Tract		Inflammations Gastrointestinal Tract		Miscellaneous	
		% Fatal	% Nonfatal	% Fatal	% Nonfatal	% Fatal	% Nonfatal	% Fatal	% Nonfatal	% Fatal	% Nonfatal	% Fatal	% Nonfatal
Coronary sclerosis * [52]	25.0	26.6	28.1	35.6	33.3	33.3	36.5	22.7	20.8	28.5	15.0	28.5	26.9
Hypertensive heart disease * [52]	22.8	24.7	34.3	34.4	30.5	45.6	40.2	18.1	19.1	7.1	25.0	42.8	40.3
Rheumatic heart disease † [87]	6.0	1.0	0.0	13.8	5.5	3.5	3.6	0.0	4.4	14.2	10.0	7.1	9.6
Syphilitic aortitis	....	3.0	6.3	11.4	6.4	8.7	6.0	0.0	0.0	14.2	0.0	7.7	1.9
Arteriolar nephrosclerosis * [52]	24.9	16.3	15.6	43.6	23.1	42.1	37.8	22.7	33.8	7.1	20.0	28.5	17.3
Benign prostatic hypertrophy † [58]	8.5	14.8	23.1	33.8	25.6	36.7	24.2	13.5	20.5	14.2	10.0	7.1	13.4
Pneumonia	....	11.3	15.6	19.5	26.8	10.5	25.6	18.1	26.4	14.2	15.0	7.1	15.3
Cirrhosis and hepatitis † [58]	5.5	98.8	93.7	12.6	6.4	14.0	10.9	9.0	1.4	14.2	30.0	28.5	42.2
Fatty liver	....	1.0	0.0	5.7	7.4	15.7	9.7	13.5	7.3	28.5	15.0	28.5	5.7
Gall bladder disease	....	16.3	28.1	10.3	14.8	12.2	13.4	13.5	4.4	7.1	15.0	14.2	9.6
Pancreatic disease † [58]	3.3	17.5	21.8	10.3	12.0	5.2	4.8	9.0	14.7	0.0	25.0	14.2	21.1
Goiter † [52]	1.1	4.1	3.1	9.1	13.8	12.2	8.5	18.1	11.7	0.0	5.0	7.1	0.0
Adrenal cortex adenoma	....	1.0	3.1	9.1	1.8	1.7	2.4	0.0	0.0	0.0	0.0	7.1	2.4
Peptic ulcer * [52]	5.0	3.0	6.3	....	....	....	....	9.0	2.9	0.0	5.0	0.0	0.0
Additional peptic ulcers † [59]	4.7	....	....	29.8	19.4	19.2	21.9	....	....	....	....	....	....

* From Cook County Hospital.
† From elsewhere.

1. *Esophageal Varices.*—In the studied material, 480 cases of esophageal varices were found of which 129 (26.8%) had a massive hemorrhage; this accounted for 19.2% of the 673 that bled (Graph 1). In 97 of the 129, hemorrhage was considered the primary cause of death (Table 2).

In the fatal cases, an anatomical diagnosis of Laennec's cirrhosis was made in 85, postnecrotic (hepatitic) cirrhosis in 10, and biliary cirrhosis in 2. Not included in this group were nine cases of cirrhosis without varices.

In the nonfatal cases, there were 24 cases of Laennec's cirrhosis, 3 of postnecrotic cirrhosis, and 2 of biliary cirrhosis, and in 3 carcinoma of the liver was found.

In the 32 cases of nonfatal hemorrhage, these were the cause of death: 23 of Laennec's cirrhosis (6 also had carcinoma of the liver, 3 tuberculosis, and 1 hemachromatosis); 3 of cardiac disease; 2 of biliary cirrhosis (1 also had choledocholithiasis and 1 carcinoma); 1 of carcinoma of the liver; 2 of toxic hepatitis; 1 of bronchopneumonia. The coexistent diseases are presented in Table 3.

2. *Gastric Ulcers.*—Of 669 cases with gastric ulcers, 87 had fatal and 108 had nonfatal gastrointestinal hemorrhage (Graph 1). Of 140 acute ulcerations, 42 bled, 14 of them fatally. Of the 529 chronic ulcers, 153 bled, 73 of them fatally (Table 4). In all, 29% of all hemorrhages and 28.1% of the fatal hemorrhages were due to gastric ulcers.

Numerous coexistent diseases were present (Table 3). The causes of death of the 108 nonfatal cases were these: 31 of carcinoma (7 lung, 4 pancreas, 2 ovary, 2 breast, 2 stomach, 2 gall bladder, 2 prostate, 2 uterus, 1 ileum, 1 bile duct, 1 eye, 1 tongue, 1 colon, 2 Hodgkin's, 1 leukemia); 27 of peritonitis (21 peptic ulcer perforations); 16 of cardiac disease (7 hypertensive heart disease, 2 rheu-

matic heart disease, 2 subacute bacterial endocarditis, 1 coronary thrombosis, 1 myocardial infarct, 1 syphilitic heart disease, 1 arteriosclerotic heart disease, 1 cor pulmonale); 9 of brain disease (6 inflammatory, 3 vascular); 9 of lung disease (7 pneumonia, 2 empyema).

3. *Duodenal Ulcers.*—Of 531 cases with duodenal ulcers, 57 had fatal and 82 others had nonfatal gastrointestinal hemorrhage (Graph). Of 75 acute ulcerations, 15 bled, one of them fatally; of 456 chronic ulcers, 124 bled, 56 of them fatally (Table 4). In all, 20.7% of all hemorrhages and 18.4% of the fatal hemorrhages were due to duodenal ulcers.

In 82 nonfatal cases these were the causes of death: 18 of cardiac disease (7 hypertensive heart disease, 3 cor pulmonale, 3 myocardial infarcts, 1 subacute bacterial endocarditis, 1 arteriosclerotic heart disease, 1 rheumatic heart disease, 1 aortic stenosis, and 1 coronary thrombosis); 15 of carcinoma (3 biliary ducts, 3 lung, 2 pancreas, 2 liver, 2 uterus, 1 sigmoid, 1 prostate, and 1 bone); 13 of peritonitis (9 perforated ulcers, 3 ruptured appendix, and 1 ruptured diverticulum of colon); 12 of brain disease (7 inflammatory and 5 vascular); 11 of lung-chest disease (6 pneumonia, 4 tuberculosis, and 1 empyema); 6 of liver disease (2 postnecrotic cirrhosis, 2 acute yellow atrophy, and 2 Laennec's cirrhosis). These and other coexistent diseases are shown in Table 3.

4. *Aneurysms.*—Of 349 cases of aneurysms, 20 bled, 19 of them fatally (Graph). Aneurysms accounted for 3% of all hemorrhages and 6.1% of the fatal hemorrhages.

The aneurysm in all but two of the fatal cases was in the aorta; in the other two it was in the hepatic and splenic arteries. There were 13 males and 7 whites. Four were 40 years or younger; 14 were between 41 and 60; 2 were past 60. Rupture into the esophagus occurred in 14 of the fatal cases.

5. *Carcinomas.*—A. Gastrointestinal (Except ampulla of Vater): Of 1341 necropsies of carcinoma of the gastrointestinal tract, 74 bled, 17 of them fatally (Graph). Carcinoma accounted for 6.9% of all hemorrhages and 4.1% of the fatal hemorrhages. There were 230 of the esophagus; 5 bled, 3 of them fatally. Of 597 carcinomas of the stomach, 47 bled, 13 of them fatally. There were 29 of the small bowel; 3 bled; none of them bled fatally. There were 495 of the large bowel; 19 bled, 1 of them fatally. Of the fatal cases, 13 were in the stomach, 3 in the esophagus, and 1 in the colon. Of those in the stomach 10 were in men and 8 were in whites; 1 case was under 40; six were between 41 and 60, and 6 were older. Analysis of the 57 cases of nonfatal hemorrhage is presented in Table 5.

B. Head of Pancreas and Ampulla of Vater: Of 112 cases of carcinoma of the head of the pancreas, 14 bled, 4 of them fatally. Three were white and three were women; two were between 41 and 60, and two were older. Of the 10 nonfatal bleeding cases, 8 were white and 6 were female; 3 were between 41 and 60, 1 was younger, and 6 were older.

Bleeding occurred in 2 out of 23 cases of carcinoma of the ampulla of Vater, 1 of which was fatal.

Certain coexistent diseases were more prevalent than others (Table 3). Of 68 nonfatal cases, the causes of death were 65 of carcinoma (33 stomach, 17 colon, 10 pancreas, 2 esophagus, 2 duodenum, and 1 ampulla of Vater); 2 of peritonitis (1 perforated diverticulitis and 1 perforated appendicitis); 1 of pneumococcic meningitis.

6. *Inflammations.*—In this series, inflammations of the gastrointestinal tract were present in 658 instances of which 34 bled, 14 of them fatally. There were 117 cases of esophagitis; 5 bled, 1 of them fatally. Of 184 cases of gastritis, 7 bled, 5 of them fatally. Of 88 cases of ulcerative colitis, 7 bled, 2 of them fatally. There were 66 cases of diverticulitis; 4 bled, 2 of them fatally. Two bled, none fatally, in six cases of gastric syphilis. Of 11 cases of jejunal ulcer, 4 bled, 1 of them fatally. Ileal ulcers were found in 51 cases; 2 of which bled, none fatally. Three of 135 cases with gastric erosions bled fatally.

In 20 nonfatal cases, these were the causes of death: 5 of carcinoma; 5 of peritonitis; 3 of liver disease; 2 of cardiac disease. Other coexistent diseases are shown in Table 3.

7. Miscellaneous.—Of the remaining 66 cases with various diagnoses, bleeding was fatal in 14.

of esophageal ulcers, and in one case each of marginal ulcer and postradiation colitis. Nonfatal hemorrhage occurred in two cases of periarteritis nodosa and in one case each of uremia, subacute bacterial endocarditis, infarction of the stomach, uterine carcinoma invading the sigmoid, and sarcoma invading the

TABLE 4.—*Relative Occurrence of Three Hundred Thirty-Four Acute and Chronic Peptic Ulcers Causing Massive Hemorrhage in the Stomach and Duodenum*

	No. of Cases	Age			Sex*		Race†	
		0-40	41-60	61 and Over	Male	Female	White	Negro
Fatal hemorrhage, 144 cases								
Total gastric ulcers	87	3	43	41	71	16	69	18
Acute	14	1	6	7	10	4	9	5
Chronic	73	2	37	34	61	12	60	13
Total duodenal ulcers	57	7	23	27	49	8	44	13
Acute	1	1	0	0	1	0	1	0
Chronic	56	6	23	27	48	8	43	13
Total fatal cases	144	10	66	68	120	24	113	31
Nonfatal hemorrhage, 190 cases								
Total gastric ulcers	108	13	44	51	82	26	84	24
Acute	28	10	7	11	20	8	24	4
Chronic	80	3	37	40	62	18	60	20
Total duodenal ulcers	82	10	36	36	62	20	54	28
Acute	14	4	6	4	12	2	8	6
Chronic	68	6	30	22	50	18	46	22
Total nonfatal cases	190	23	80	87	144	46	138	52
Total of all hemorrhages of gastric and duodenal peptic ulcers	334	33	146	155	264	70	251	83

* 42% of admissions are females.
† 42% of admissions are Negroes.

TABLE 5.—*Age, Sex, and Race of Fifty-Seven Cases of Nonfatal Massive Hemorrhage of Carcinoma of the Gastrointestinal Tract* *

	Age			Sex		Race	
Carcinoma	0-40	41-60	61 and Over	Male	Female	White	Negro
Esophagus	0	2	0	2	0	2	0
Stomach	1	18	15	33	1	28	6
Small bowel	0	1	2	2	1	2	1
Colon	2	6	10	9	9	10	8
Totals	3	27	27	46	11	42	15

* Does not include 1 at ampulla of Vater.

There were 25 instances in which no lesion or condition could be determined after a complete autopsy to account for the bleeding; in 4 it was fatal. Of 14 cases bleeding with blood dyscrasias, 2 bled fatally. In nine cases, fatal in one, diseases of the liver but no varices were found. In three cases, fatal in two, varices were present but no bleeding point. In two cases, fatal hemorrhage occurred from spontaneous tears of the gastric mucosa following emesis; this occurred also in two cases

duodenum. Of 242 cases with benign tumors of the gastrointestinal tract, 2 bled, none fatally.

COMMENTS

Sex.—In the total series of 673 cases, 511 were men and 162 were women, a ratio of 3.2:1. In 310 fatal cases, 250 were men and 60 were women, a ratio of 4.2:1. Of the 363 nonfatal cases, 261 were men and 102 were women, a ratio of 2.5:1. The combined ratios in favor of men is greater than the admission

ratio. This observation is in general agreement with others.§ Most clinical reviews show an even greater male preponderance: usually four or five to one, favoring the male.‖ Males account for 58% of admissions to the Cook County Hospital.

Race.—There were 503 white and 170 Negro patients, a ratio of 2.9:1. Of the fatal cases, 246 were white, a ratio of 3.8:1. Of the nonfatal cases, 257 were white, a ratio of 2.4:1. At the Cook County Hospital 58% of the patients are of the white race. The frequency and mortality of hemorrhage favors the white race.

TABLE 6.—*Age Distribution in Six Hundred Seventy-Three Cases of Massive Gastrointestinal Hemorrhage*

	Fatal		Nonfatal	
Decade	No.	%	No.	%
0-10	7	2.2	14	3.8
11-20	2	0.6	5	1.3
21-30	5	1.6	11	3.0
31-40	34	10.9	38	10.4
41-50	70	22.7	73	20.1
51-60	95	30.6	85	23.4
61-70	65	20.6	86	23.7
71-80	27	8.7	40	11.0
81-90	5	1.6	10	2.7
91-100	0	0.0	1	0.3
Totals	310		363	

Age.—Fatal hemorrhage occurred in all decades. In 48 cases (15.3% of all fatal cases) under 40 years, bleeding was fatal (Table 6). No great difference between fatal and nonfatal hemorrhage was present in any particular decade. The finding of 30.9% of fatal hemorrhages and 37.7% of nonfatal hemorrhages in patients over 60 years of age would seem to indicate that age alone was not the only factor involved in the prognosis of the hemorrhage. There was no great variation in the age incidence among different anatomical diagnoses.

Anatomical Findings.—This study reveals a marked discrepancy of the incidence of accepted causes of gastrointestinal hemorrhage (Graph). Before concluding that these differences are due entirely to the type of patients attracted to any hospital reporting this data, one would have to correlate their clinical impressions with the morphologic events available in their institution and ascertainable only by autopsy. For example, though it has been previously reported from this hospital[81] that clinically cirrhosis accounted for 9.8% of upper gastrointestinal hemorrhage, the autopsy incidence was 19.2% of all hemorrhages.

The difficulties in establishing the correct diagnosis are many; one of these is the simultaneous presence of multiple lesions, each potentially a bleeding site. Cirrhosis was present in 6 of the 87 fatal cases of gastric ulcer and in 6 of the 57 fatal cases of duodenal ulcer. Ratnoff and Patek,[65] in 386 cases of Laennec's cirrhosis found an incidence of peptic ulcers of 3.6%. Lipp and Lipsitz,[48] after finding that peptic ulcer had an incidence of 11.5% in portal cirrhosis and 14.3% in biliary cirrhosis, concluded that in the presence of obvious cirrhosis with massive hemorrhage, it is not justifiable to assign automatically the cause of bleeding to a ruptured varix. The incidence of cirrhosis without varices [88] and series of varices without cirrhosis [28] at necropsy have been reported.

The frequency in which the autopsy findings do not substantiate the clinical diagnosis has been noted. Chalmers [11] and others,¶ reported that in 24% of his 101 fatal bleeding cases the bleeding was not suspected during life; also, 20 cases from that series had multiple possible sources for hemorrhage.

Coexistent Diseases.—Certain diseases are encountered in a higher than usual incidence when bleeding gastrointestinal lesions are present (Table 3). While it has long been recognized that the aging process materially influences the mortality of gastrointestinal hemorrhage, not all these diseases have their greatest frequency in the geriatric patient. The usual explanations offered for the clinical observation of increased mortality with increasing years have been that (1) the chronicity of an ulcerating lesion makes healing

§ References 2, 11, 29, 37, and 62.

‖ References 2, 16, 17, 47, 78, and 83.

¶ References 15 and 20.

more difficult; (2) the degenerative changes in vessels around ulcers prevent retraction and contraction; (3) the compensation for blood loss is less effective with advancing years; (4) the nutritional deficiencies are more frequent in the older age group.

The presence of coronary sclerosis in bleeding patients creates a dangerous combination.# The factors concerned in disproportion between supply and demand to the heart muscle are (1) occlusive disease of the coronaries; (2) fall in blood pressure; (3) lowered oxygen-carrying capacity of the blood, and (4) increased work demands.[60] Of the occlusive coronary artery disease, arteriosclerosis is the cause in over 90%. Thus, in a bleeding case all factors could be present to give maximum cardiac embarrassment. Paterson [60] states that acute functional insufficiency of the coronary circulation from a sudden increase in the nutritional demands of the heart in the presence of severe arteriosclerotic stenosis is far more common than generally realized. He feels that shock or hemorrhage may be the precipitating factor. In a review of 103 cases of acute hemorrhage (95 of the gastrointestinal tract), Master [50] found 57% showed clinical and electrocardiographic evidence of coronary insufficiency up to the severity of actual infarction.

Wartman [86] described intramural capillary rupture of the coronary vessels with hematoma formation. Current investigators feel arteriosclerosis is an instability of the serum cholesterol being deposited at the points of pre-existent vascular injury. It is felt that intramural arterial hemorrhage is frequently the "previous injury" and that this is caused by high intracapillary pressure from arterial hypertension. Coronary thrombosis is more common in hypertensives than in nonhypertensives.* Kinney and Mallory [44] and others [2] noted the occurrence of coronary thrombosis precipitated by gastroduodenal hemorrhage in vessels which showed no previous arterial disease.

Whereas it is recognized that definitive surgery in a bleeding cardiac patient is fraught with increased risk, there is much to suggest that the indication for surgery is even greater.

Arteriolar nephrosclerosis is found with high frequency in patients with gastrointestinal hemorrhage. This is frequently associated with hypertension.[56] The kidney so afflicted may function adequately until such crises as prolonged shock, blood transfusion incompatability or, dehydration and then its limited "reserve" would predispose to renal insufficiency. This would be aggravated still further by the biochemical alterations seen with gastrointestinal hemorrhage; namely, hyperazotemia, hypochloremia, and alkalosis.[13]

The presence of an increased incidence of prostatic hypertrophy may also put greater tax on already limited kidney function. The frequency of pneumonia and syphilitic lesions reflects their occurrence before the days of antibiotics.

Experimentally, blood lost from the femoral artery is better tolerated than a comparable amount lost from the hepatic.[46] Owing to shock alone, portal vein oxygen drops from 75% to as low as 20%. The oxygen tension of the hepatic artery is decreased by escape of blood from ulceration just a few centimeters away. This is aggravated by spasm of the vessel, which leads first to hypoxia and finally anoxia. The lethal effect of anoxia of the liver and its role in irreversible hemorrhagic shock is known.†

Though the pancreas is not dependent upon a single vessel, the question arises as to how susceptible is the pancreas to reduced oxygen tension. Experimentally, pancreatitis can be more readily produced when the vessels are ligated.

Further discussion on coexistent diseases will follow with each anatomical cause for bleeding.

Esophageal Varices.—The over-all incidence of varices of the esophagus was 1.7%.

References 50 and 51.

* References 60 and 86.

† References 26 and 22.

The incidence reported elsewhere varies from 0.3 to 8%.‡

Of the 97 fatal cases with esophageal varices, 47.4% had ascites; of the 32 nonfatal cases, 62.5% had ascites. Icterus was present in 31.9% of the fatal cases and 31.3% of the nonfatal ones.

The cause of the varices was cirrhosis in all but three cases; in two of these, chronic portal vein obstruction was present, and in one no cause was found. The high incidence of benign prostatic hypertrophy may be explained in part by the liver disease. The incidence of gall bladder disease suggests an increased association of these two diseases. Similarly, the increased occurrence of pancreatic disease was expected in the presence of high incidence of liver disease. The frequency of chronic cholecystitis and cholelithiasis in diabetics has been shown.[7] There was no marked increase in the incidence of goiters as Moon[55] states occurs with cirrhosis.

Peptic Ulcers.—The finding of 1200 active ulcers gives an autopsy incidence of 4.23%. At the Philadelphia General Hospital it is 2.75%.[29] Gordon and Manning[29] collected from the literature 197,556 autopsies on persons with peptic ulcers in this country in the past 60 years; the over-all incidence of peptic ulcer was 3.51%. Ivy[38] states that from 5 to 12% of the population became afflicted with ulcers.

In 27.1% of the total number of cases of peptic ulcer, Collins[16] reviewed 2620 clinically proved cases of ulcers and found 3.8% bleeding. Others § found 10% in smaller clinical series.

Peptic ulcers accounted for 50.9% of all cases of gastrointestinal bleeding. This is at variance with the customary incidence cited on the basis of clinical evidence (Table 7).

In the total series, more gastric than duodenal ulcers were found. While at difference with clinical impressions, it is comparable to other necropsy series.‖ The clinical impression of the more frequent occurrence of duodenal ulcer varies from 2:1[1] to 9:1.[16] The greater hazard of bleeding from gastric ulcers has been noted in both clinical ¶ and necropsy reports.#

Gastric Ulcers (Peptic).—There were 669 cases of active acute and chronic gastric ulcers found, indicating a necropsy incidence of 2.4%.

Fatal bleeding here favored the male 4:1. Of all gastric ulcers, 20% were acute ulcers. Compared to acute duodenal ulcers, these occurred twice as often, and bled three times as frequently, and with greater mortality. This has been noted by others.[10]

TABLE 7.—*Clinical Incidence of Peptic Ulcer as a Cause of Gastrointestinal Bleeding*

Name	Year	No. Bleeding Cases	Per Cent Peptic Ulcer
Allen [1]	1937	231*	63.6
Brown [9]	1950	224*	76.0
Bulmer [10]	1927	526	89.0
Collins [16]	1939	141*	70.9
Costello [17]	1949	300	68.0
Rivers [63]	1931	199	78.3
Sullen et al.[81]	1949	305*	75.0
Thompson [88]	1946	264	42.2

* Upper gastrointestinal tract.

The increased incidence of coronary sclerosis with gastric ulcer has been noted by others; Gordon and Manning[29] found "cardiac lesions" in 37.4% of their 558 peptic ulcers and 40.3% in those cases in which an ulcer and cirrhosis were present.

The finding of more than usual amount of hypertensive heart disease is important from other aspects. Wartman and Hellerstein[87] noted nearly three-fourths of their hypertensive hearts had coronary artery disease and evidence of myocardial disease.

In the fatal cases, arteriolar nephrosclerosis occurred twice as often as otherwise. Pancreatic disease occurred with increased frequency; this has been observed similarly by others.[71]

The finding of increased number of goiters and adrenal cortex adenomas may bare

‡ References 28 and 58.

§ References 1 and 89.

‖ References 6, 11, 14, 29, 58, and 62.

¶ References 25, 42, 53, and 77.

References 6, 10, 11, and 62.

greater importance in light of the theory brought forth by Gray and others.[31] They feel that in times of stress the stomach may act as an endocrine organ working as part of a hypothalamic-pituitary-adrenal-gastric axis. The interrelationship between goiters and forms of heart disease is controversial.[35]

The finding of additional peptic ulcers in nearly one-third of the fatal cases points out the frequency with which multiple lesions occur. This amount was greater than a report [29] from the Philadelphia General Hospital where 4.7% of the patients with gastric ulcer at autopsy had a duodenal ulcer present also.

Duodenal Ulcers.—The incidence of active duodenal ulcer at autopsy was 1.9%. Other reports in the last century vary from 0.4 to 5.75%.* Fatal bleeding here favored the male 6:1.

Coronary sclerosis, hypertensive heart disease, and arteriolar nephrosclerosis occurred more frequently with duodenal ulcer than without them.

The usual incidence of cirrhosis at autopsy varies from 1.86% [23] to 7.1%.[11] Gordon and Manning [29] found 11.9% in 558 cases of peptic ulcers; some types of "liver damage" was found in 58% of their peptic ulcers, compared to 35% found at routine autopsy. Fatty liver occurred three times as frequently here as it did in the gastric ulcers.

Aneurysms.—The necropsy incidence of aneurysm of the aorta was 1.2%. It is significant that this diagnosis was not even considered in the previous clinical resumé (Table 1).

In 1950, a total of 111 cases of nontraumatic aortic perforations rupturing into the gastrointestinal tract were collected from the literature; [4] others have been added.[30] This paper will add 22 cases: 17 aortic aneurysms rupturing into the gastrointestinal tract, 4 carcinomas of the esophagus eroding the aorta, and 1 diverticulitis of the colon with abscess eroding the aorta. Aneurysms of the hepatic artery rupturing into the gall bladder have been reported; [20] one such case is included here.

Of the 17 aortic aneurysms, 16 were syphilitic and 1 was arteriosclerotic. Turner [54] reviewed 4322 cases of syphilis and found aneurysm or aortitis in 7.65%.

Carcinomas.—The incidence of carcinoma of the alimentary tract (exclusive of the oral cavity and pharynx) was 4.8%; esophagus, 0.8%; stomach, 2.1%; small bowel, 0.1%; large bowel, 1.8%. Gastric carcinoma had an incidence of hemorrhage in 7.8%. The necropsy incidence for carcinoma of the head of the pancreas was 0.4%. The low incidence of hemorrhage in carcinoma has been noted in both clinical and necropsy series.†

There was an increased incidence of hypertensive heart disease, hepatobiliary disease, pancreatic disease, and goitrous lesions.

Inflammations.—Gastritis was the cause of 1.6% of all fatal hemorrhages; clinically, it was found in 5.1% of the cases (Table 1). In Chalmers and others,[11] 101 fatal cases of gastrointestinal hemorrhage, the diagnosis was made clinically in 40 cases; at autopsy of these same cases it was found in 2. Appreciating the difficulties of substantiating the clinical diagnosis, we agree with Crohn [7] that gastritis is not a frequent cause of hemorrhage; this is contrary to other opinions.‡

Esophagitis, diverticulitis, and ulcerative colitis rarely were the cause of massive hemorrhage. No one died of hemorrhage of gastric syphilis.

Jejunal ulcers were considered for the most part peptic in origin. Ileal ulcers represent a combination of tuberculous, typhoid, and regional ileitis and lymphoid ulceration.

There were three fatal cases from multiple erosions of the gastric mucosa; all patients were of the white race, and two were males. There were no intracranial lesions found.§ Irritations of the vagus have been known to produce these.[67] Friedman [27] produced these lesions by injection of pilocarpine, epinephrine, and thyroid substance.

* References 14, 53, and 61.

† References 9, 10, 11, 17, 67, and 68.

‡ References 5, 12, 17, and 59.

§ References 19 and 80.

The few cases of coexistent diseases do not lend themselves to conclusions on this topic.

Miscellaneous.—There were 25 cases of "parenchymatous hemorrhage"; the intact mucosa and submucosa contained large quantities of blood, and there was no systemic or local disease present upon gross and microscopic examination. There are numerous accounts of unknown bleeding.‖ Chalmers, and others,[11] reported seven fatal hemorrhages where the site of bleeding could not be determined. Many explanations have been offered for such bleeding, for example, gastritis,[74] duodenitis,[69] chronic aspirin toxicity,[70] alcoholic debauches and vomiting,[49] and a hepatosplenic syndrome.[66] Korner [45] recognized the entity of parenchymatous hemorrhage and stated it was an actual permeability of the vessels without microscopic evidence. He would not include those cases where tabes, scurvy, sepsis, or hunger had been present.

In 1929, Mallory and Weiss [49] described the occurrence of fatal hemorrhage following laceration of the cardiac orifice of the stomach due to vomiting. Two such cases are in this series: one, a 26-year-old Negro woman who developed fatal hematemesis after vomiting from acute alcoholism; the other, a 58-year-old white man with multiple lacerations of the stomach after the ingestion of ipecac.

The infrequency of massive hemorrhage from a marginal ulcer was surprising.

Although the literature contained numerous accounts ¶ of bleeding from small bowel tumors, we found none recorded.

There were 29 cases of diaphragmatic hernia, without bleeding. This lesion, which is recognized with greater facility in the living, rarely bleeds fatally [13]; gross bleeding,# chronic anemias,[75] and other complications have been described.

In this series, hemorrhage did not occur in any case [7] of chronic pancreatitis of pancreaticolithiasis as has been reported elsewhere [24]; two cases have been seen since that time.

In this group there was an increased incidence of hypertensive heart disease and liver disease. In the four fatal cases of parenchymatous hemorrhage, a hypertensive heart was present. There were no peptic ulcers found in these miscellaneous cases. Wood [90] cautions that in bleeding occurring after splenectomy for essential thrombocytopenia a peptic ulcer must be excluded.

‖ References 39, 40, 41, 54, 79, 83, and 85.

¶ References 43, 63, 64, and 76.

References 8, 33, and 70.

SUMMARY

In a study to determine the anatomical cause of massive gastrointestinal hemorrhage, the protocols of 28,400 consecutive autopsies at the Cook County Hospital were reviewed. Massive gastrointestinal hemorrhage was found in 673 cases; in 310 cases it was the cause of death, and in 363 cases it was contributory or incidental to it.

Fatal hemorrhage occurred most frequently from esophageal varices. Gastric ulcers, the second most frequent cause, occurred more frequently and bled more frequently, and with greater severity than duodenal ulcers which were the third largest group. The fourth was arterial aneurysms rupturing into the gastrointestinal tract. Hemorrhage associated with carcinoma comprised the fifth group. Fatal hemorrhage in diverticulitis, colitis, or gastritis was rare.

In 25 cases no morphological cause could be ascribed to the bleeding. These were considered parenchymatous in nature and were fatal in four instances.

Fatal bleeding was found in all ages, including the newborn period. The greatest incidence of fatal hemorrhage occurred during the fifth, sixth, and seventh decades of life. In the decades marked by the occurrence of degenerative diseases, the incidence of fatal hemorrhage was less than that of nonfatal hemorrhage.

There was a 4 to 1 predilection of the male sex and white race for fatal hemorrhage, suggesting an endocrine factor.

The following coexistent diseases were found present in an increased frequency:

coronary sclerosis, hypertensive heart disease, syphilitic aortitis, arteriolar nephrosclerosis, benign prostatic hypertrophy, hepatic and pancreatic lesions, goiter, and adrenal cortex adenoma. In the cases of bleeding peptic ulcers, a high incidence of multiple peptic ulcers was found. The high incidence of certain coexistent diseases suggests that some conventional contraindications to surgical hemostasis may actually be indications. The effect of hypotension and blood loss on these organs whose physiological reserve is limited may be actually the cause of death and not the hemorrhage itself.

The diagnosis of the causes of gastrointestinal hemorrhage utilizing present clinical methods is strikingly inaccurate if a correlation with anatomical findings is attempted. Improved methods of the detection of the site of bleeding in the living patients are needed.

Dr. Hans Popper, Director, Department of Pathology, Cook County Hospital, assisted in the preparation of this paper.

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Latex and Thorium Dioxide (Thorotrast) in Treatment of Aneurysms

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The treatment of aneurysms has tried the ingenuity and skill of surgeons from the earliest of recorded medical experience. In the third century, Antyllus [1] recommended ligation of the proximal and distal artery, followed by opening and evacuation of the sac. In 1786, John Hunter [1] advised proximal ligation well above the diseased portion of the artery to prevent erosion of the wall and hemorrhage, an operation which he performed successfully on many occasions.

After the monumental discoveries of Lister, treatment of these lesions was greatly enhanced, and the way prepared for the more definitive aneurysmorrhaphy techniques described in 1889 by Matas.[2] Although Carrel * wrote little on the subject of aneurysm itself, he described accurate methods for blood vessel anastomosis, demonstrated the feasibility of arterial grafting by the employment of fresh and preserved vessels and so made possible the excision of aneurysms and their replacement by preserved grafts. This method, which has been employed in the abdominal and thoracic aorta by Dubost [5] and his co-workers, by DeBakey and Cooley,[6] Lam and Aram,[7] and many other authors, is now the accepted method of treatment for most aneurysms of large vessels. Certain saccular types have also been excised by Bahnson [8] with primary suturing, a method similar to the aneurysmorrhaphy of Matas.

There can be little remaining doubt that the ideal treatment for large aortic aneurysms is excision, followed by reestablishment of the vessel continuity, through grafting or by suture anastomosis. Certain of these lesions, however, are so large, or so situated that their excision is technically not feasible without great risk to the patient. Advanced age, severe heart or kidney disease, and other general conditions may also render resection of large aneurysms impracticable. Under such circumstances, the surgeon is left with two alternatives: He may defer any definitive treatment, or he must find some less radical though less effective method of therapy.

Older procedures for the control of large aneurysms, while not curative, have produced good results in many advanced cases, with a low mortality and morbidity. Chief among these have been wrapping with polyethylene (Polythene) or Cellophane, reported by Poppe,[9] Yeager and Cowley,[10] Chunn [11] and others, and wrapping with Cellophane combined with wiring, reported by Blakemore [12] and others. Certain disadvantages of Cellophane have limited its use, particularly the development of tumors in rats in which it was used and the difficulty of securing the snug, well-fitting application of a sheet of Cellophane or polyethylene about the rounded contour of an aneurysm. Experimental studies by Woodhall [13] demonstrated that liquid latex applied on arterial walls produced a considerable degree of fibrosis.

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Latex for these experiments was supplied by the United States Rubber Co.

From the Department of Surgery, The George Washington University School of Medicine.

* References 3 and 4.

Ideally, a substance most suitable for the wrapping of these lesions should be nontoxic and should be applicable in a liquid form and develop into a solid, tough resilient coat within a short time after exposure to the air. It should adhere readily to the vessel wall, protecting the aneurysm itself. In order to find such a material, animal experiments were carried out in the surgical laboratories of the George Washington University School of Medicine.

Before carrying out experiments to determine what substance if any might be applied over the surface of aneurysms to strengthen their walls and stimulate fibrous tissue formation, it first was necessary to find a satisfactory method for the production of aneurysms in experimental animals. Following a suggestion of Dr. Robert Hill,† who had noticed aneurysm formation in the arteries of autopsied patients treated with intra-arterial mustard, nitrogen mustard solutions were injected into the adventitia and media of dogs' aortas. When 10 mg. of nitrogen mustard was injected, necrosis of the arterial wall resulted, and the dogs usually died of aortic rupture within a few days. With doses of 5 mg., definite dilatation of the aortic wall resulted, with rupture in all but one surviving dog within 8 to 17 days.[14]

With the use of such experimental aneurysms, an attempt was then made to find a liquid or semiliquid substance which could be applied over the aortic wall to cause fibrosis and strengthen the vessel. In some instances the aneurysm was produced first and the supporting substance applied at a later stage, but since several animals died of aortic rupture before the material could

† Personal communication to the authors.

Fig. 1.—*A*, appearance of thorium dioxide and latex 13 days after its application over an experimental aneurysm. *B*, same; internal surface.

be applied, in later animals the nitrogen mustard was injected and the vessel supported in the same procedure.

RESULTS

One dog with an aneurysm to which 10 mg. of liquid nylon was applied died of aortic thrombosis on the fifth postoperative day.

One animal treated with liquid Cellophane died of unknown cause on the fifth postoperative day.

One animal treated with Fiberglas died of aortic rupture on the 24th postoperative day.

In four animals, cutis skin grafts were applied over the aneurysms. Three died of aortic rupture; one died of unknown cause.

In 11 animals, split-thickness skin grafts were applied over the aneurysms with plasma as an adhering agent. Five of the dogs died of aortic rupture; four died of distemper; one died of chylothorax, and one died of empyema.

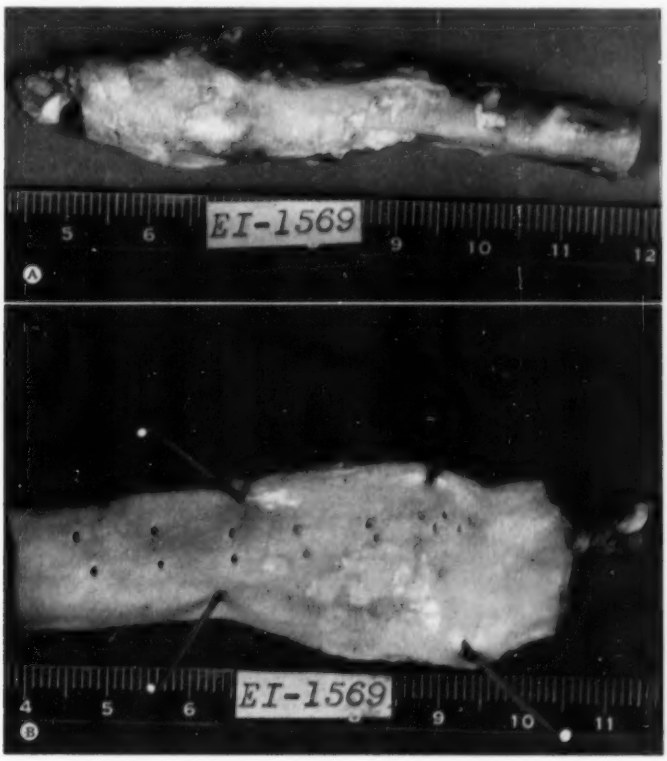
In one dog the aneurysm was covered with a suspension of asbestos, but the animal died on the third postoperative day, of aortic rupture.

In 13 dogs a form of liquid latex, which became a paste on sterilization, was applied and precipitated by the aid of calcium sulfate. This resulted in poor adhesion of the latex to the vessel wall. Eleven of the 13 animals died of aortic rupture. At autopsy, little or no adhesion of the latex to the vessel wall was found.

In nine dogs liquid latex sterilized by autoclaving was applied over the aneurysm wall. Three animals survived; three died of aortic rupture, and three died of other causes.

In 20 dogs, after injection of 5 mg. of nitrogen mustard, the aortic wall was painted

Fig. 2.—*A*, thorium dioxide and latex 39 days after its application over an experimental aneurysm. *B*, same; internal surface.



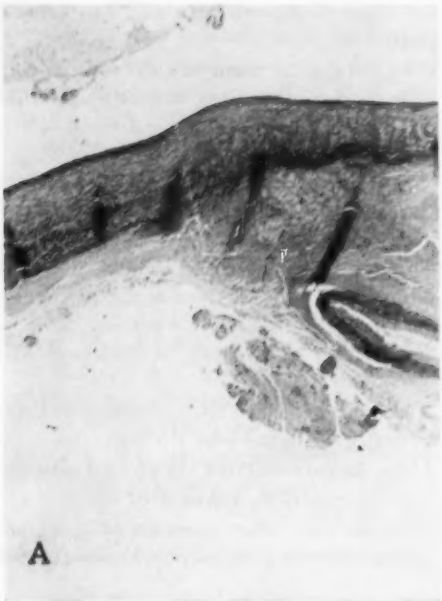


Fig. 3 *A*.—Extensive fibrous reaction at site of application of latex and thorium dioxide over a dog's aorta. Thickness of fibrosis is shown by comparison with the normal adventitia.

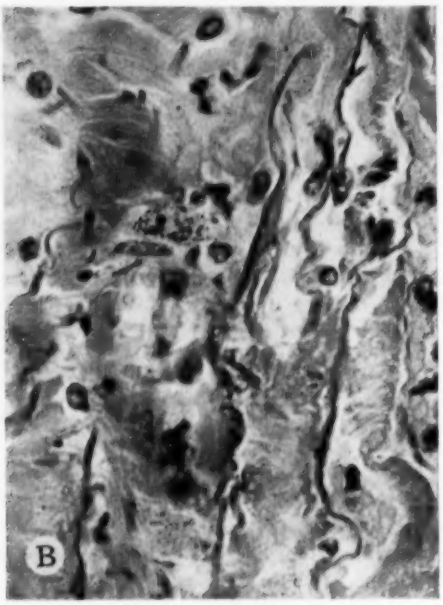


Fig. 3 *B*.—Thorium dioxide particles in reactive fibrous tissue.

with lactic acid 0.5%, followed by a thin coat of a mixture of thorium dioxide (Thorotrast) (one part) and liquid latex (eight parts). This was allowed to dry with the aid of a light current of air and was then covered with several successive layers of the latex-thorium dioxide mixture. Calcium sulfate was then powdered lightly over the vessel to cause firm coagulation of the outer coat. Fourteen of the 20 animals survived for periods up to eight months; 3 died of distemper; 2 died of unknown cause, but only 1 died of aortic rupture (Figs. 1 and 2).

Thorium dioxide is known to produce dense fibrosis when allowed to escape accidentally into perivascular tissues in the performance of arteriography. This fibrosis

Results of Wrapping of Experimental Aneurysms

	Total Dogs	Survived	Died, Aortic Rupture	Died Distemper	Died Other Causes
Nitrogen mustard					
Aneurysm controls					
10 mg. mustard	9	0	3 (33%)	3	3
5 mg. mustard	5	1	3 (60%)	..	1
Treated aneurysms					
Cellophane	1	0	.....	..	1
Liquid nylon	1	0	.....	..	1 (aortic thrombosis)
Fiberglas	1	0	1 (100%)	..	..
Cutis graft	4	0	3 (75%)	..	1
Split-thickness graft	11	0	5 (45%)	4	2
Asbestos	1	0	1 (100%)	..	..
Paste latex	13	1	11 (84%)	..	1 (aortic thrombosis)
Liquid latex	9	3 (33%)	3 (33%)	..	3
Latex thorium dioxide	20	14 (70%)	1 (5%)	3	2
Total	75				

increases with the passage of time, and, although instances have been reported in which it appeared to be carcinogenic, this effect has usually followed its injection into the blood stream, and then only after the passage of many years. In the advanced stages of arterial disease for which it is proposed here, this does not appear to be a valid contraindication to its use.

Extensive fibrotic thickening at the site of application of latex and Thorotrast over the adventitia of a dog's aorta is shown in Figure *3A* and Thorotrast granules in the fibrous tissue in Figure *3B*.

Based upon the experimental data described above, latex and thorium dioxide were applied to aortic aneurysms in three clinical cases.

REPORT OF CASES

CASE 1.—A 66-year-old white man was hospitalized on Feb. 8, 1954. He stated his present illness had begun approximately two years previously when he noted lethargy, low back pain, and aching across the smaller portion of his back. One year ago, he noted a mass just to the left of the umbilicus which in the interim had not increased noticeably in size. Six weeks prior to admission he awoke one morning with a distinct aphasia, with numbness on the right side of his face and numbness of his right hand. There was evidence of weakness and the sensation, and the aphasia had cleared up by evening.

Physical Examination: The patient was a fairly well developed, well nourished, 66-year-old man. Blood pressure 180/120. Pulse 96. Heart sounds were of fair quality. There appeared to be some left ventricular hypertrophy. On abdominal palpation there was a pulsating mass arising to the left of the umbilicus and extending upward and laterally from the right on both sides. There was no thrill or bruit on palpation or auscultation. Extremities—varicosities of both lower legs. Femoral and iliac vessels were both easily palpable and pulsated normally. The dorsalis pedis could not be detected on either foot. Oscillometric examination revealed good pulsations in both upper and lower legs.

X-Ray Studies: A chest plate on Feb. 19, 1954, showed a density in the left upper field and a density in the left lower field within the heart shadow, thought to represent an aneurysm of the aorta. An aortogram on Feb. 9, 1954, showed marked irregular dilatation of the abdominal aorta at its junction with the thoracic aorta. There was a second aneurysm just below the first, extending to the first sacral segment. The latter one was only partially filled by the contrast medium, apparently owing to thrombosis and clots. The common iliac arteries and their branches were not filled with dye.

Laboratory Data: On Feb. 9, 1954, cardiolipin test was negative for syphilis. Urinalysis was essentially negative. CBC: WBC 9,500; RBC 4,200,000; Hemoglobin 13 gm. Hematocrit 41; differential: neutrophiles 60, lymphocytes 40. MCV 91 and MCC 30. On Feb. 12, 1954, PSP total dye excreted 38%. On Feb. 15, 1954, urea nitrogen 15.8%.

EKG: On Feb. 12, 1954, an electrocardiogram showed the following: (1) vertical heart sign; (2) sinus arrhythmia, and (3) essentially normal. Repeat EKG on March 7, 1954, showed a sinus rhythm with frequent premature beats which seem to be ventricular, probably nodal, and premature auricular contraction.

Course in Hospital: On Feb. 10, 1954, a lumbar aortogram was done and revealed marked involvement of the aorta by at least two aneurysms. The renal arteries were involved in the lower aneurysm. This is in the left. On Feb. 25, 1954, with the patient under general anesthesia, the abdominal aneurysm was exposed and dissected free. The renal arteries were seen to come from the lower portion of the upper aneurysm. The lower abdominal lesion was then covered with latex and Thorotrast since the entire lesion was nonresectable. Although his first postoperative day was quite satisfactory, during his entire postoperative course he was confused and complained of lower abdominal pain. Severe pain developed on Feb. 28, 1954. On March 6, 1954, he was found unconscious and somewhat cyanotic. On the following day he began to have liquid bowel movements which soon became bloody. Abdominal cramps became more severe, and the abdomen became distended. On the following day his blood pressure continued to drop in spite of intravenous fluids and blood. He died March 9, 1954. Autopsy revealed mesenteric thrombosis.

CASE 2.—A 52-year-old housewife was admitted to George Washington University Hospital for the first time on Sept. 2, 1952, with a complaint of paroxysmal nocturnal dyspnea. The patient stated that nine days prior to admission she was awakened from sleep by a tight, girdle-like sensation around her chest and found herself gasping for breath. There was a moderate cough, productive of white frothy sputum. Her past history was not remarkable except for gastric resection performed in 1949 and a hysterectomy for a uterine fibroid in 1940.

Physical Examination: The patient was a moderately obese, well developed woman in no evident dyspnea or other distress. Her blood pressure was 130/90, respirations 24, pulse 80. Chest expansion was equal, and the left lung was clear. There were increased spoken and whispered voice sounds over the apex of the right lung and middle and upper

portions of the right middle lobe. A grade III, rough, blowing, systolic murmur was heard most clearly over the aortic area, and a grade II murmur heard over the pulmonic area. The aortic murmur could be heard radiating to the left side of the neck. There was no tracheal tug.

X-Ray Examination: Roentgenograms of the chest showed an anterior, superior mediastinal tumor projecting into the lung fields on the right side. An electrocardiogram on Sept. 3, 1952, revealed frequent unifocal, premature ventricular contractions. ST and T-wave changes were characteristic of digitalis effect.

Cardiolipin serological tests for syphilis were 4 plus (2 units), but the Kahn was negative. Other laboratory tests were within the normal range, except that the urine was loaded with leucocytes. To determine the nature of the mediastinal mass, exploratory thoracotomy was advised.

Operation: With the patient under satisfactory anesthesia, the chest was entered by resecting the right fourth rib, subperiosteally. All structures within the pleural cavity on the right side appeared to be normal, with the exception of the aorta. The vena cava showed no evidence of obstruction and the azygos vein was of normal size. A large aneurysm commenced at the base of the aorta as it ascended from the heart and involved the entire arch of the aorta past the great vessels. The origin of the innominate artery could be seen underneath the aneurysm and the latter continued well past the take-off of this vessel. As the aorta swung into its descending portion, it could be palpated and was felt to decrease in size. There was no pulsation within the enlarged vessel. The pleura was opened over the top of the aneurysm opposite the superior vena cava, and the latter was dissected free from the dome of the lesion so that the most superior and arching portion of the aneurysm was freed. A piece of Cellophane containing diacetyl phosphate was placed over the aneurysm and held in place by suturing the pleura together at the corners and tacking the Cellophane to the edges of the pleura. The lung was then re-expanded and the chest closed. The postoperative course was uneventful, and she was discharged on Sept. 17, 1952.

Second Admission: On May 12, 1953, the patient was readmitted to the hospital because of three episodes of paroxysmal dyspnea similar to that for which she was admitted originally. The last attack was extremely severe. She had been restricted also in her physical activity because of shortness of breath during the day but had had no anginal pain or chest pain of any description.

Physical examination revealed a well-healed thoracotomy scar. There was a grade I aortic systolic murmur but no cardiac enlargement, and the lungs were clear.

X-Ray Studies: Roentgenograms of the chest revealed the aneurysm as previously noted without essential change.

Because of the recurrence of symptoms, reoperation seemed advisable, to attempt replacement of the aneurysm by a graft.

Operation: On May 20, 1953, with the patient under satisfactory anesthesia, a transverse incision was made beginning near the right anterior axillary fold extending across along the second rib over the sternum onto the second rib of the left side nearly to the left axillary fold. Bilateral subperiosteal resection of the second ribs was performed and the right pleural cavity opened. The incision was carried toward the midline and the aneurysm located. The sternum was then exposed by cutting the attachments of the pectoralis major and with the fingers of the operator under the sternum and over the aneurysm, first the anterior, then the posterior tables of the sternum were fractured. Many adhesions were found from the old operative procedure where the Cellophane had been placed. Complete mobilization was carried out, but it was found that although the aneurysm was somewhat saccular, it was also generally enlarged in a fusiform dilatation. As it was soft and friable in many areas, resection was not technically feasible. Therefore, a mixture of sterile, liquid latex containing one part Thorotrast to four parts of latex was applied over the aneurysm in several layers, drying each layer under a current of warm air. When completed, a firm, elastic coating of latex was visible on all exposed surfaces of the aneurysm. The layers of the chest wall were then closed with running, chromic, and interrupted 0000 black nonabsorbable (silk) sutures.

Postoperative Course: After this procedure the patient was relatively asymptomatic and had no further attacks of paroxysmal dyspnea. She was readmitted on May 7, 1954, because of recurrent epistaxis, and the bleeding was controlled by cauterization with silver nitrate and packing. She was readmitted in June, 1954, because of hemoptysis productive of blood-streaked, dark brown sputum. This had occurred three days before admission during a paroxysm of coughing. She had had no recurrence of the chest pain or orthopnea and had otherwise been perfectly well since her last admission. X-rays of the chest showed no change in the aneurysm, and she was discharged nine days after admission. In July, 1954, she died suddenly, apparently owing to rupture of the aneurysm. A postmortem examination could not be obtained, since the patient died at home.

Case 3.—A 56-year-old white man was hospitalized on Feb. 19, 1953, because of a large aneurysm of the abdominal aorta. In May, 1952, a 3½-ton truck ran over his abdomen, resulting in a fracture, dislocation of the left sacroiliac joint, and separation of the symphysis pubis. In October, 1952, a fusion

of the left sacroiliac joint had been performed because of continued pain in this region. At this time an abdominal aneurysm was detected by x-ray examination of the abdomen.

Physical examination on admission revealed a well developed, fairly well nourished white man, ambulatory with the use of a cane. General physical examination was within the limits of normal except for the following findings: There was a large pulsating mass in the lower midabdominal region which corresponded to the x-ray evidence of an abdominal aortic aneurysm. There was a left indirect inguinal hernia; there was a well-healed scar in the region of the left sacroiliac joint from the previous fusion.

Laboratory Work: Tests on admission resulted in the following: normal hemogram, negative Kahn, negative urinalysis; total proteins 7.2%, albumin 4.0% and globulin 3.1%; NPN was 38 mg.%. X-ray examination of the heart and lungs revealed a mild cardiomegaly. X-ray examination of the pelvis revealed an old healed fracture of the left ilium with a considerable amount of sclerosis at the fracture site. Abdominal films revealed a calcified area anterior to the second, third, and fourth lumbar vertebrae which outlined a mass measuring 14×11 cm. in size which was typical of aneurysm of the abdominal aorta. EKG was reported as within the limits of normal.

Addendum to Interim Summary dated April 17, 1953: Exploratory laparotomy for resection of the aneurysm was undertaken. At operation a saccular aneurysm measuring 7×5 in. was found involving the right and left renal arteries. The aneurysm was felt to be unsuitable for a grafting procedure, and it was painted with latex and Thorotrast and covered with nonreactive Cellophane. Postoperatively the patient developed a wound infection which eventually was controlled, with the use of erythromycin. The patient was discharged on Aug. 8, 1953.

In July, 1954, the patient had no pain or other symptoms referable to the aneurysm.

SUMMARY AND CONCLUSIONS

Experimental aneurysms were produced in 75 dogs by the injection of nitrogen mustard into the adventitia and media of the thoracic aorta. When untreated and unsupported, all surviving dogs with such experimental aneurysms except one died of rupture of the vessel. Various substances including liquid nylon, Cellophane, Fiberglas, cutis skin grafts, split-thickness skin grafts, asbestos, liquid latex and latex mixed with thorium dioxide (Thorotrast) were then applied over the aneurysms in separate groups of dogs, to determine which substance, if any, would support the aneurysms sufficiently to prevent their rupture. The following two effects of such substances were sought: (1) the mechanical supporting of the aneurysm and (2) the stimulation of fibrous tissue formation. The best results were obtained in aneurysms treated with a mixture of liquid latex and thorium dioxide. Of 20 dogs so treated, 14 survived; 3 died of distemper; 2 died of unknown cause, but only 1 died of rupture of the aneurysm. The next best results were obtained in animals treated with latex alone. Of 9 such animals, 3 survived. Three died of other causes, and three died of rupture of the aneurysms.

On the basis of these experiments, latex and Thorotrast have been applied to three large, otherwise inoperable, aortic aneurysms in humans.

In Case 1, the patient died in the immediate postoperative period owing to mesenteric thrombosis. There was no latex or Thorotrast in contact with any of the mesenteric vessels, and the cause of the thrombosis was felt to be traction on the mesentery by the delivery of the small bowel outside the abdomen during dissection of the aneurysm.

In Case 2, the patient survived for two years, but died of rupture of a large aneurysm of the aortic arch. Her pain was greatly relieved, although this may have been due in part to decompression of the lesion by transection of the upper sternum in the operative approach. In view of the size of the aneurysm, however, the latex and thorium dioxide may well have prolonged the life of this patient.

The patient in Case 3 is surviving two and a half years after application of the latex and thorium dioxide to a huge abdominal aneurysm. Pain has been relieved, and he now has no symptoms referable to the lesion.

As a result of these experiments and clinical cases, we believe that a latex thorium dioxide mixture is a satisfactory coating for the supporting of large aneurysms, which, because of their size, location, or the general condition of the patient, cannot be treated by resection.

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Scientific Exhibits

PERINEAL PROSTATECTOMY IN THREE DIMENSIONS

EDWIN DAVIS, M.D.
AND
LEROY W. LEE, M.D.
OMAHA

PERINEAL PROSTATECTOMY IN THREE DIMENSIONS and color was prepared with the purpose of demonstrating the possibility of complete hemostasis and clear visibility throughout the entire procedure, which we believe to be contrary to popular conception. The 20 representative views here presented were selected from a total of 36. The accompanying statistical charts are based upon a study of mortality, convalescence, complications, and late functional results.

For a detailed consideration of the factors which have gradually eliminated or minimized the recognized hazards and complications and which have transformed perineal prostatectomy into an innocuous procedure of precision, the reader is referred to "Progress in Perineal Prostatectomy: Report of 2050 Consecutive Cases," by Davis and Lee, in the January, 1955, issue of the *Journal of Urology*.

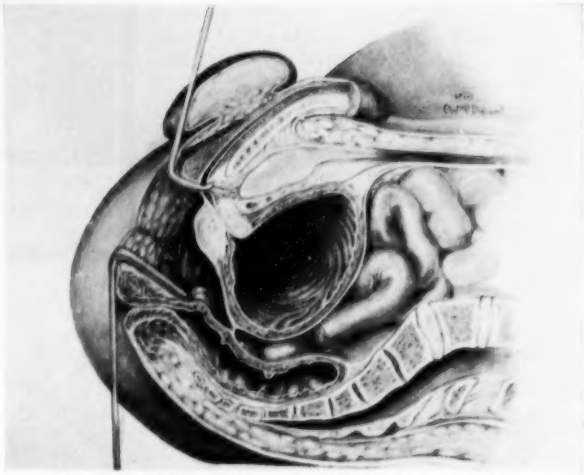
NOTE.—It is suggested that the small photographs are better viewed through a reading glass.

From the Department of Urology, University of Nebraska College of Medicine.

Shown as a Scientific Exhibit of the Section on Urology at the 103rd Annual Meeting of the American Medical Association, San Francisco, June 21-25, 1954.

Gross midline section—anatomical

SURGICAL ANATOMY



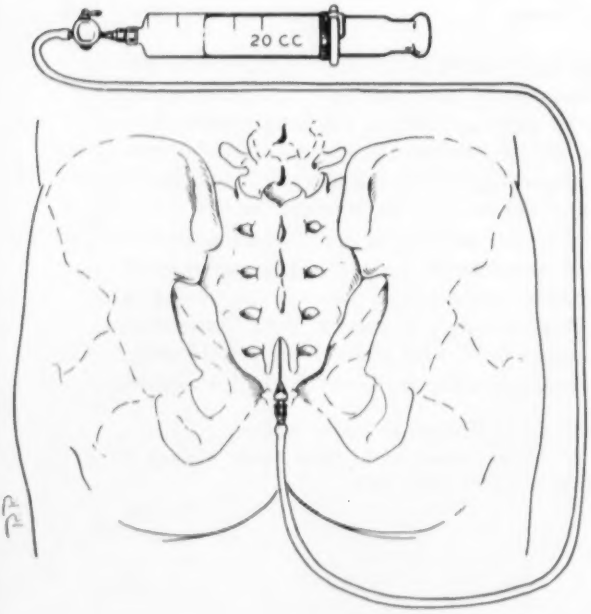
Gross midline section—perineal surgical approach

INDICATIONS FOR PERINEAL PROSTATECTOMY

1. Moderate to large benign prostatic hyperplasia with obstructing symptoms.
2. Prostatic calculi with obstructing prostate.
3. Early carcinoma of the prostate.

ANESTHESIA

Continuous caudal, using 0.5% solution of hexylcaine (Cyclaine) hydrochloride, 80 cc. initial dose, supplemented as necessary.



Diagrammatic illustration of caudal anatomy and continuous caudal technique.

Perineal prostatectomy patients.....2,050
Total deaths.....60 Mortality rate.....2.9%

Analysis as to Cause of Death

Deaths directly related to prostatic surgery.....24

- Sepsis 15
- Hemorrhage 1
- Delayed Hemorrhage 5
- Uremia 3

Deaths of doubtful relationship to prostatic surgery.....30

- Cerebral accident 8
- Coronary thrombosis 6
- Pulmonary embolus 15
- Bronchopneumonia 1

Deaths, wholly unrelated to prostatic surgery.....6

- Cerebroarteriosclerosis 1
- Femoral embolus 1
- Acute hemorrhagic gastritis..... 1
- Perforation of colon 1
- Parkinsonism 1
- Intestinal obstruction 1

Total deaths 60

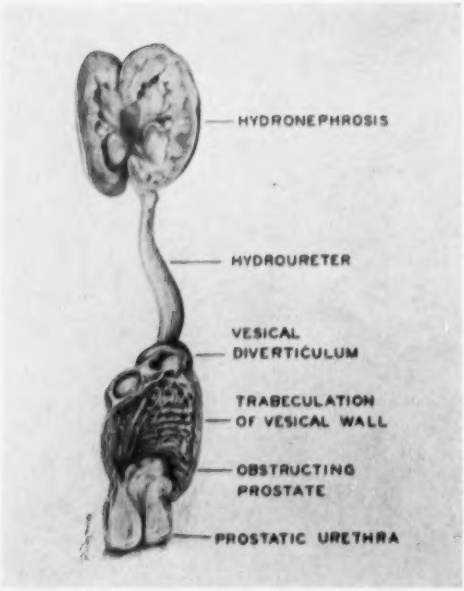
Reported Mortality for Prostate Surgery in Various Clinics

		Number of Patients	% Mortality
Perineal prostatectomy			
Hugh Young	Baltimore	1,000	3.2
Daniel Higbee	Denver	572	1.7
Davis and Lee	Omaha	2,050	2.9
Suprapubic prostatectomy			
Hunt and Walters	Rochester, Minn.	1,000	5.4
Leon Herman	Philadelphia	750	4.0
Alexander Randall	Philadelphia	340	6.3
Freyer	England	1,550	5.3
Retropubic prostatectomy			
Miller	England	757	4.5
Jacobs	Glasgow	500	6.0
Newman	Sidney, Australia	103	8.0
Transurethral prostatectomy			
Reed Nesbit	Ann Arbor	2,425	1.8
Creevy	Minneapolis	1,500	1.2
Randall	Philadelphia	315	2.2
Kretschmer	Chicago	551	3.2
Alcock	Iowa City	999	4.6
Lee	Omaha	993	1.9

POSTOPERATIVE CONVALESCENCE

1. Up first or second postoperative day
2. Sutures, drain, and catheter out seventh postoperative day
3. Discharge from hospital eighth to twelfth postoperative day

END-RESULT OF NEGLECT OF THE OBSTRUCTING PROSTATE



HYDRONEPHOSIS

UREMIA

DEATH

Urinary tract from patient with fatal obstructing prostate.

END-RESULT OF PERINEAL PROSTATECTOMY

1. Life
2. Comfort
3. Anatomical restoration of urinary tract
4. Functional restoration of urinary tract

ONE-YEAR FOLLOW-UP QUESTIONNAIRE

100 Consecutive Patients Who Had Perineal Prostatectomy
Result (Patient's Own Opinion)

Well 89%
Improved 10%
Unimproved 1%

Vasectomy (epididymitis prophylaxis); loop of vas dissected free for excision

Continuous caudal anesthesia; needle introduced; equipment attached

Perineal elevator in position on operating table. Note shoulder straps for maintaining position

Transurethral prostatic tractor introduced

Blunt dissection of ischiorectal space

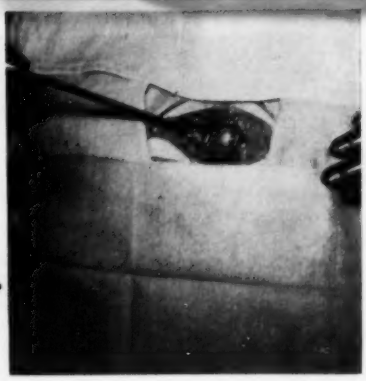
Bifid retractor stretching central perineal tendon

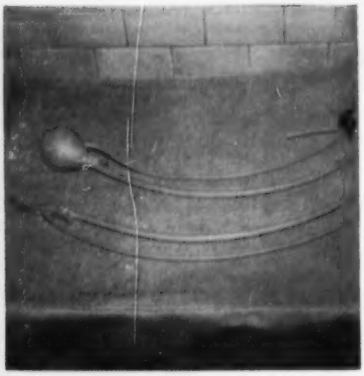
Denonvilliers' fascia covering posterior surface of prostate

Weighted posterior retractor with assorted blades

Inverted V incision through posterior prostatic capsule into prostatic urethra

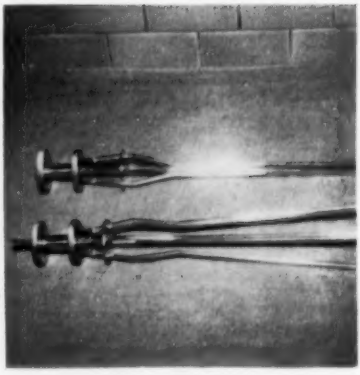
Prostatic lobe forceps grasping hyperplastic lobes

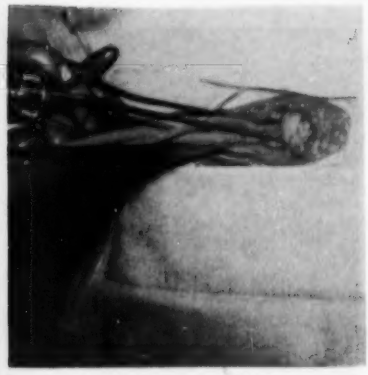




Perineal hemostatic traction bag

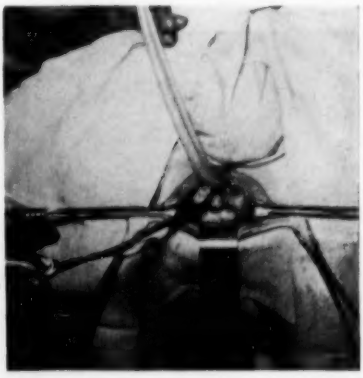
"Umbrella" — four-pronged guide for introducing bag





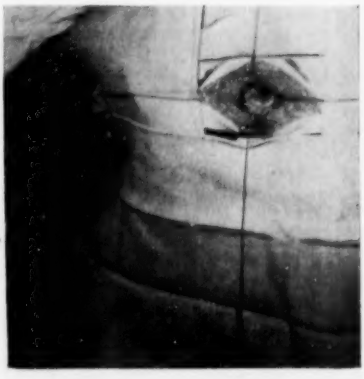
Introduction of hemostatic traction bag into bladder, with use of sponge forceps and "umbrella" guide

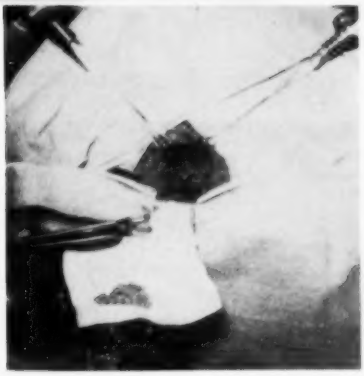
Traction on hemostatic bag; enucleation continued



Enucleation completed. Note clean vesical neck

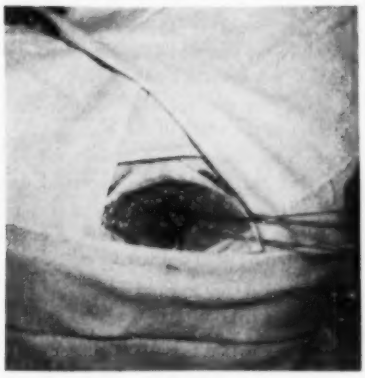
Plastic closure, showing four quadrant sutures, constricting vesical orifice and providing hemostasis





Vesical calculi removed

Closure of capsule completed; V-flap replaced and anatomic relations restored; catheter invisible



Levator ani closure; sutures placed, not tied.

Irrigation returned clear; hemostasis satisfactory

Case Reports

SUBMUCOUS LIPOMA OF STOMACH

Case Report and Review of the Literature

MELVIN A. CASSEL, M.D.
and
JOSEPH B. GUCCIONE, M.D., St. Louis

MASSIVE hemorrhage from an unknown source in the gastrointestinal tract remains one of the most perplexing problems in surgery. Lacking a history of preexisting ulcer or gastritis, negative physical findings, a questionable roentgenogram, the following case report illustrates such a problem with a most unusual lesion as the bleeding source.

J. P., a 49-year-old white man, was admitted to Faith Hospital on April 25, 1953, with a complaint of tarry stools of five days' duration. The black stools had increased in frequency and volume in the 48 hours prior to admission. Except for the gradual progressive weakness, there were no associated symptoms.

There was nothing in the past history suggestive of gastrointestinal disease. However, for the past two years this patient had been under medical care for hypertension and episodes of auricular fibrillation.

During this time he had received various drugs including phenobarbital, veratrum viride (Vertavis), mannitol hexanitrate (Nitranitol), alkavervir (Veriloid), and conquinone (Quinidine) and, recently hydralazine (Apresoline) and a brand of hexamethonium chloride (Methium). His blood pressure decreased from 230/120 to 170/100 under a dosage of 250 mg. of Methium four times daily.

On admission, the patient appeared pale, anxious, and acutely ill. His blood pressure was 180/90; the heart was enlarged to palpation; rhythm was regular, and the rate was 100/min. The abdomen was soft and moderately distended. There were no areas of tenderness or muscle guard. Intestinal peristaltic sounds were increased. There were no other positive physical findings.

The RBC on admission were 2,270,000; WBC 9,300, and hemoglobin 44%. The differential count was reported as 4% stab cells, 65% segmented cells, and 31% lymphocytes. Platelets were 215,000 and prothrombin time 20 seconds. Urinalysis was negative for albumin and sugar; the microscopic examination revealed amorphous phosphate crystals and occasional epithelial cells.

An upper gastrointestinal series was done on the morning of admission and was reported as showing no evidence of organic lesion involving the esophagus. Palpation of the abdomen was not done because of the clinical history, but good roentgenograms were obtained after the administration of barium by mouth, and no definite evidence of organic lesion was seen in the stomach or duodenum. Slight irregularity of contour of the barium-filled stomach was noted on the lesser curvature side high up near the junction with the esophagus, best observed in the upright position (Fig. 1).

The chest film was reported normal, except for enlargement of the heart to the left.

The electrocardiogram was interpreted as showing auricular fibrillation and myocardial damage.

Course in hospital: The morning after admission, while walking to the toilet, the patient fainted and fell to the floor. The resident was called and found the patient pulseless and confused. Supportive treatment, including 2 units of blood was given, and the patient recovered from shock.

Later in the day, he vomited approximately 250 cc. of dark blood, some of which was clotted; this was the first episode of hematemesis. Three additional units of blood were given in the next 24 hours without further episodes.

However, on the third day of hospitalization, there was a massive hematemesis, amounting to about 1000 cc. of blood. His blood pressure dropped to 110/40; his pulse was 140. Shock therapy was instituted and blood transfusions were given continuously until the patient was brought to the operating room, about three hours later. At this time, his RBC count was 3,080,000 and hemoglobin 60%.

From the Surgical Service of Faith Hospital.

Fig. 1.—Roentgenogram showing the filling defect high on the lesser curvature.

In view of the life-threatening hemorrhage, an exploratory laparotomy was decided upon. On exposure, the stomach and bowel were found filled with blood. There was no external evidence of scar in the region of the pylorus or duodenum, and both organs were freely movable. On palpation of the stomach, a firm globular mass was found on the anterosuperior portion of the lesser curvature. The mass was about the size of a golf ball and freely movable beneath the serosa. The stomach wall was incised over this prominence and, on retraction of the serosa, an encapsulated yellow fatty tumor was found between the serous and mucosal layers. The inner portion of the tumor was adherent to the mucous membrane, and a segment of this layer had to be resected with the tumor in order to remove the mass.

On gross inspection of the specimen, there was a deep, well-defined ulcer, 1 cm. in diameter, in the center of the attached mucosa.

The gastrotomy wound was closed in layers and the abdomen closed without drainage. The patient's condition was good throughout the procedure.

His postoperative course was smooth with removal of the Levin tube after 48 hours, and the patient placed on oral feedings at this time. He was discharged on the eighth postoperative day with a moderate anemia—RBC 3,500,000 and hemoglobin 78%.

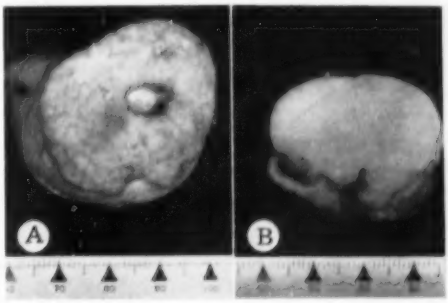
The surgical specimen was examined by Dr. John Bauer and described as follows: "Gross description: The specimen consists of an oval piece of gastric wall which measures 4 × 3 cm. The center of the mucosa is occupied by an ulcer which measures 1 × 0.7 cm. The ulcer perforated the mucosa and the base is formed by a lipoma which is incompletely capped by gastric mucosa. The lipoma measures 4 × 3.5 × 1.5 cm. and is situated in the submucosa below the muscularis mucosae and above the main layer (Fig. 2). Microscopic findings: Section shows the gastric mucosa interrupted by an ulcer, the base of which is formed by a zone of peptic digestion. This zone rests for the most part on mature adipose tissue which shows inflammatory infiltration and evidence of mucoid degeneration. Close to the surface of the ulcer is a blood vessel which is occluded by a thrombus. There is also some blood at the base of the ulcer.

"Histopathological diagnosis: Stomach, chronic peptic ulcer with thrombosed artery in base. Perforation of ulcer into submucosal lipoma."

COMMENT

The massive hematemesis of bright red blood and clots placed the bleeding point in the stomach, esophagus, or duodenum. The roentgenogram demonstrating the irregular filling defect high on the lesser curvature (Fig. 1) served only to add confusion and was not seriously considered as the cause of hemorrhage. Only in retrospect did it appear significant. Rather, it was felt that the antihypertensive drugs which this patient had been taking for the past two years might have resulted in one or more large superficial ulcers or in hypertrophic gastritis, either of which would have been consistent with the clinical picture. Despite this lack of a definite diagnosis, the patient's poor condition and repeated severe hemorrhages demanded prompt exploration.

Fig. 2.—Showing the depth of the ulcer in the gastric mucosa and the firm attachment of the submucous lipoma to the mucous membrane.



It was felt that the high location of the ulcer on the lesser curvature and its intimate relationship with the underlying lipoma justified the decision to perform a wedge resection rather than the more formidable subtotal resection.

It was also apparent after examination of the ulcer and the size of the artery at its base that recurrent hemorrhage would have been certain under medical management.

GENERAL OBSERVATIONS

Benign tumors of the stomach comprise about 1% of all tumors of this viscus; lipomas make up but a small percentage of this group. The fact that these tumors may be confused clinically and radiographically with malignant and inflammatory lesions adds to their surgical importance. According to Moore,[1] benign tumors of the stomach present certain findings which serve to differentiate them from other lesions: The filling defect is circumscribed and has a regular curvature; the rugae surrounding a benign tumor are more nearly normal in arrangement and distribution; there is little or no disturbance in peristalsis and retention is uncommon except when the lesion is at or near the pylorus; there is no niche, incisure, or other evidence of spasm.

While the roentgenologic examination may be helpful, the diagnosis of the majority of submucous lipomas has been made at surgery or autopsy.

In 1941, Rumold[2] reviewed all cases of submucous lipomas reported since 1835. This comprised a total of 33 cases, including his own presentation. Since then, an additional 10 cases have appeared in the literature.*

It is also possible that the incidence of these tumors may be greater than the reports indicate, since it is reasonable to assume that many gastric lipomas never produce symptoms unless their size, position, or superimposed ulceration create complications.

Prominent among the symptoms of the reported cases is bleeding; approximately one-third of the cases presented this symptom. Other common complaints were epigastric distress, anorexia, nausea, and vomiting.

Obstruction of the pylorus, resulting from the prolapse of a pedunculated lipoma, has also been reported.[4]

Anemia and weakness due to melena is found in cases in which ulceration or erosion of the projecting portion of the tumor has occurred.

Because of the frequent association of ulceration with the lipoma, it would appear that these tumors exert some necrotizing effect on the mucosa or that peristalsis over the projecting mucous membrane leads to mechanical erosion. In either event, gross bleeding of a recurrent nature would seem likely, inasmuch as the fatty tissue around the bleeding vessel offers little hemostatic effect.

Essentially the symptoms and signs are those of any upper gastrointestinal lesion and there is no pathognomonic complaint.

The pathogenesis of these benign tumors is unknown. Some authors[10] suggest the congenital origin of these tumors, arising from an anlage separated during fetal life. Others believe that chronic irritation, either chemical or physical, gives rise to low-grade inflammation of the stomach wall.

Minnes and Geschicter[10] conclude that although chronic irritation and inflammation may be contributory factors in the production of gastric tumors, they are by no means essential causative agents.

Grossly the tissue of the lipoma has a soft, greasy, yellowish lobulated appearance not unlike lipomas found elsewhere in the body. Microscopically, also, they show typical adipose structure.

Treatment of the lipoma consists of resection of the tumor with its attached mucosa or serosa. Recurrences have not been reported following this simple procedure.

* References 3 through 9.

SUMMARY

A case of submucosal lipoma of the stomach with ulceration and massive hemorrhage is reported. These tumors are uncommon. The symptoms, signs, and roentgenologic findings may be confused with those of malignant or benign lesions of the stomach. Treatment consists of simple excision of the tumor.

2801 N. Taylor (15).

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Special Reports

ABSTRACTS of Papers Presented at Association of Surgeons of Great Britain and Ireland, The London Sectional Meeting of the American College of Surgeons, and the Special Meeting of the International Society of Surgery in Lyon.

INTRODUCTION

In May, 1954, approximately 500 Fellows of the American College of Surgeons attended a sectional meeting of the College in London in the halls of the Royal Academy of Surgeons of England at Lincoln Inn Fields. Prior to this, the American College Fellows were guests of the Association of Surgeons of Great Britain and Ireland at their annual meeting in Leeds, and after the London meeting the Fellows attended special meetings of the International Society of Surgery in Paris, Lyon, and Strasbourg. Many of the Fellows attended special clinics at the Universities of Glasgow and Edinburgh in Scotland prior to the Leeds meeting.

The character and content of the presentations were so interesting and informative that it seemed greatly worth while to record and publicize them. Abstracts of the presentation at the American College meeting in London will be found particularly interesting because of the active participation of members of the Royal College in each presentation. This was true not only at the scientific meetings but at the panel discussions at each noonday luncheon as well. Abstracts of these presentations and summaries of the Hunterian Lecture on exophthalmos by Dr. Howard C. Naffziger and the Moynihan Lecture on pancreatitis by Dr. Walter C. MacKenzie were so well presented in a publication in *The Lancet* that the editors take pleasure in republishing that report. This was made possible through the courtesy of the editor of *The Lancet*, Dr. T. F. Fox.

Abstracts of the papers presented at the Association of Surgeons of Great Britain and Ireland appeared in their program, and these are reprinted with permission of the President of the Association, Mr. Digby Chamberlain, of Leeds.

The two-day meeting in Paris consisted for the most part of attendance at operative and demonstrative clinics, but a brochure giving the viewpoint of the Parisian surgeons on the treatment of various surgical lesions was presented to each Fellow. Permission for publication of this material in the A. M. A. Archives of Surgery has been obtained from Professors Louis Bazy and René Leriche.

At Lyon, abstracts of presentations made before this group at demonstrations and operative clinics were included in the booklet which gave the program of clinics and demonstrations; these are republished with permission of Prof. Pierre Mallet-Guy.

Professors Fontaine and Weiss arranged two days of operative clinics and demonstrations at Strasbourg. Much to my regret, I was unable to attend this meeting. Those who could and did were enthusiastic about the fine surgical work which they saw in progress there.

It was a rewarding trip—not only from the scientific standpoint, but for the opportunity to see our colleagues abroad in action in the operating theater and on the speaker's platform; both were interesting, stimulating, and informative. The time and care taken in preparing programs and selection of topics for discussions were evident in every city and every hospital visited, and the rewards of this cooperative effort by surgeons, their associates and assistants, working with local administrators, were greatly appreciated by all of their American and Canadian visitors.

Waltman Walters, M.D.

ASSOCIATION OF SURGEONS OF GREAT BRITAIN AND IRELAND

The following abstracts of papers are taken, with slight editorial changes, from the program of the Annual Meeting of the Association of Surgeons of Great Britain and Ireland, Leeds, England, May 13-15, 1954.

From Discussion on Hiatus Hernia.

P. R. Allison: The morbid anatomy of hiatus hernia is described, and important etiological factors that contribute either to the production of the hernia or the production of symptoms are emphasized, with some remarks on differential diagnosis. Associated anomalies in the upper abdomen are mentioned. The combination of hiatus hernia with an esophagus partially lined with gastric mucous membrane is considered in relation to symptoms and the necessity for accurate preoperative assessment. The selection of patients for operation, the preoperative preparation and operative technique, and the results of operations during 1951, 1952, and 1953 are presented.

STUART W. HARRINGTON: Herniation of the abdominal viscera through the esophageal hiatus is the most common of the various kinds of diaphragmatic hernia.

These hiatal hernias are of considerable general interest because of their relatively frequent occurrence; the multiplicity of the factors involved in their causation; the variation of the relationship between the defective esophageal hiatus and the esophagus, which results in different types of hiatal hernia; the progressive character of their development, which results in varied and complex symptoms that often simulate those of other organic disease; the occurrence of complications such as incarceration, obstruction, and ulceration of the stomach and esophagus, incompetence of the cardia, esophagitis, and stricture of the esophagus, and their treatment, which may be conservative if the hernia is small and uncomplicated and causes only mild symptoms but which requires surgical intervention if the symptoms are severe and are not relieved by conservative measures.

The technical surgical considerations of hiatal hernia depend upon the type of hernia which is encountered. I do not believe that there is any one method or technique by which all the different types of hiatal hernia can be satisfactorily repaired surgically. The technical factors depend upon the size and location of the defective esophageal hiatus as well as on the position and function of the cardia and the lower part of the esophagus. The important considerations of surgical repair are replacement of the herniated viscera below the diaphragm, reconstruction of the esophageal hiatus at a higher level on the esophagus, and in some cases reimplantation of the esophagus at a higher level in the diaphragm as well as establishment of normal function of the stomach and esophagus.

Of the 605 patients with various types of diaphragmatic hernia upon whom I have operated, 489 had the esophageal hiatal type. In the series of the esophageal hiatal type, there were 13 recurrences, in 6 of which there was recurrence of symptoms requiring second operations. In the remaining 7 recurrences there were few or no subjective symptoms and the evidence of recurrence was on a roentgenologic basis. There were 6 operative deaths, giving a mortality rate of 1.2%.

N. R. BARRETT: My remarks concern sliding hiatal hernia; the other varieties of hernia which pass through the esophageal hiatus are not so common in my experience. Sliding hernia must be clearly separated from congenital short esophagus because, although the pathological changes which result from both can be similar, the surgical treatment is different.

Sliding hiatal hernia matters for one reason only; it may lead to reflux esophagitis, and this disease, or its complications, can cause crippling illness. A sliding hiatal hernia is a hernia *en glissard*; the contents of the hernia itself never strangulate, and I know of no record of a case of strangulation of a loop of bowel in the sac. I have never found the sac to contain anything at operation. The reason that these patients develop esophagitis is that the cardiac sphincter has become inefficient, and the whole object of surgical treatment is to restore the function of this barrier. It is not in any way concerned with repairing a hernia to "prevent the risk of strangulation."

Reflux esophagitis produces a variety of changes in the lower esophagus, which call for different surgical treatments according to their degree and severity. It has been my practice to divide patients into the following categories:

(1) Those who have symptoms without evidence of active esophagitis. These patients can usually be controlled by conservative means, of which postural treatment is the basis.

(2) Patients suffering from attacks of uncomplicated esophagitis. Excellent clinical results can be obtained by restoring the function of the cardiac valve surgically.

(3) Patients whose principal disability is vomiting or hemorrhage from the esophagus. These can sometimes be managed by postural treatment but respond well to surgical measures which restore the function of the cardia. In my experience they do not always get better as a result of operations designed to diminish the digestive secretions of the stomach.

(4) Once a stricture has developed between the esophagus and the stomach the problem becomes complicated. Several varieties of stricture occur. Medical treatment is of no permanent avail for any of them. The surgeon must overcome the obstruction and prevent subsequent reflux of acid pepsin into the gullet. To achieve this purpose the relative merits of a variety of operations will be discussed.

From Discussion on Ulcerative Colitis.

FRED RANKIN: My part in this symposium necessarily has to do with the surgical approach in the treatment of chronic ulcerative colitis. Confusion which continues over the etiological factor or factors involved in the production of this ailment multiplies the difficulties of arriving at a conclusion as to adequate and standardized therapeutic measures. Since more than three-fourths of the cases are probably due to an organism or a combination of organisms, and since this group or a larger group is subject to remissions and therefore to some amelioration of symptoms by medical measures, one cannot assume that radical surgery,

which is essential in a high percentage of cases, can be instituted very early in the disease.

Chronic ulcerative colitis starts low in the rectum in about 95% of the cases and advances progressively around the circumference of the bowel. Segmental chronic ulcerative colitis occurs in a very small group of cases and is amenable to segmental resection and restoration of the bowel continuity.

Another important factor in influencing early and radical surgery in the treatment of chronic ulcerative colitis is its relationship to malignancy. Long series of cases appearing in the literature definitely buttress the thesis that the fear of malignancy is a reason for surgery as well as obstruction, hemorrhage, perforation, and intractability to medical management.

Long-standing chronic cases of chronic ulcerative colitis do develop pseudopolyposis which subsequently becomes malignant. It is impossible to arrive at an accurate percentage because of the difference in numbers of the cases studied. My own belief and experience is that about 2.5% of ulcerative colitis cases develop polyposis or pseudopolyposis. This percentage, as I reported in 1939, is lower than the general average. Dukes and his colleagues, reporting 63 cases treated surgically, found 7 cases developing carcinoma, making an incidence percentage of 11.1. Somewhere between these two figures seems to be the approximately correct one.

One may say then, that ulcerative colitis, in the chronic form, with its tendency to undergo remissions and in a great many instances to permit the patient to resume a gainful occupation, does, in a sufficiently large number of cases, develop complications or intractability to medical treatment which demand surgical aid. I believe that in the chronic ulcerative colitis the crux of the situation is to select the optimum time when, after a number of so-called cures by medical management, the disease becomes stationary or progressively worse and at that time do a total colectomy and ileostomy either in one, two, or three stages. The rectum should be removed, either as a separate stage or as a part of the primary stage, because of the reflex by which its salvage seems to influence the action of the ileostomy. The trend definitely, I think, is toward this type of procedure and I believe toward instituting it earlier in the disease than formerly was done. The mortality is below 5%; the operation is formidable but not too difficult for a skilled surgeon to accomplish in a reasonable time, and, after all, the elimination of the entire colon, which is now functionless, useless, and a so-called "lead-pipe" colon, seems to be the answer in the largest percentage of cases. About 15% of the whole series of cases will come to surgery, and I think the surgeon's acumen in selecting the proper time to institute surgery is probably one of the most important factors in the successful treatment of this unique malady. There is a small group of cases which develop rapidly and present a fulminating picture of overwhelming infection and which because of loss of blood and body fluids rapidly undermine the normal physiology to a point where, if it is not quickly scotched, the condition becomes irreversible. I have found that in this particular group of cases the institution of an ileostomy without exploration unquestionably is a lifesaving procedure.

J. C. Goligher: *Primary Excisional Surgery in the Treatment of Ulcerative Colitis.*

The reasons for the abandonment of ileostomy alone as the initial procedure in the treatment of ulcerative colitis are discussed, and the results obtained in 74 cases treated by primary excisional surgery at St. Mark's Hospital are described. This series includes no patients suffering from massive hemorrhage or perforation, but it does comprise 8 very ill cases in which the operation was undertaken as a lifesaving measure. There were only 2 deaths, and it is felt that these results substantiate the claims for primary excision.

Three forms of primary excisional surgery were employed: ileostomy with primary subtotal colectomy (followed by excision of the rectum at a later stage), ileostomy with proctocolectomy, and subtotal colectomy with ileorectal anastomosis. The relative merits of these three techniques are examined. The number of cases treated by the last method is too small and the period of follow-up too short to permit of an accurate appraisal of its place in the surgical treatment of colitis, but it is considered that for the majority of cases coming to surgery an ileostomy will always be indispensable. The importance of efficient ileostomy management for the well-being of these patients cannot be too strongly stressed. The first essential for success in this matter is a well formed ileostomy, and a few of the more important points in the operative technique are emphasized. The second requirement is a well designed adhesive type of ileostomy bag, the fitting and care of which should be clearly explained to the patient. Reference is made to a recently written "Booklet of Advice for Ileostomy Patients," which has been found very helpful at St. Mark's Hospital.

From Discussion on Exophthalmos.

Harvey Jackson:

Terminology

Diagnosis: Implications of unilateral and bilateral disturbance

Summation of causative pathologies

Duration of protrusion with lack of realization on the part of the patient

Variations in degree—amount and mechanisms involved

Investigations

(1) Radiology

(2) Hematology

(3) Others

Differentiation between neoplastic and endocrine exophthalmos

Treatment

(*a*) Surgical

Indications for operation treatment

Scope of intervention

Various techniques

(*b*) Nonsurgical

ARTHUR JONES: *The Role of Radiotherapy in the Management of Exophthalmos.*

Progressive exophthalmos is believed to originate from pituitary overactivity related to, but not necessarily identical with, excessive production of thyrotropic hormone. The pathological changes in the orbits in this special ophthalmopathic variety of Graves' disease include enlargement of extraocular muscles, round cell infiltrations, and edema of the fat and connective tissue. The cellular infiltrations (which may be of an intensity resembling those seen in leukemia) are of a type known to be highly radiosensitive, while the resolution of similar chronic inflammatory reactions elsewhere is known to be favorably influenced by irradiation.

Radiation therapy may therefore be employed to attempt depression of anterior pituitary function, or to cause regression of the orbital infiltrations and edema. The natural history of progressive exophthalmos is variable, and while some cases may progress to the malignant phase, with keratitis or papilledema threatening vision others may become spontaneously arrested. An attempt will be made to assess the value of the radiotherapeutic methods against the variable natural history of the disease.

From Discussion on Duodenal Diverticula.

JOHN M. WAUGH AND EDWARD V. JOHNSTON: *Primary Diverticula of the Duodenum.*

The authors have reviewed 30 cases of primary diverticula of the duodenum in which surgical exploration was carried out at the Mayo Clinic from 1940 to 1952, inclusive.

Eight patients had more than one duodenal diverticulum; 5 patients had additional diverticula in the jejunum; and 9 of 20 patients who underwent barium enema studies had diverticulosis of the colon.

The diagnosis of symptomatic duodenal diverticula can be extremely difficult to establish and should not rest solely on an otherwise negative examination. Rather, a careful clinical history must be relied upon, in a more positive approach to the establishment of the presence of organic disease or dysfunction. A thorough trial of medical therapy should then be carried out prior to advising surgical intervention.

Surgical exploration was undertaken in only 30 of 525 cases in which duodenal diverticula were noted on the records by the clinicians during this 12-year period. In only 10 patients were the diverticula the chief indication for exploration. The results from surgical repair of the diverticula in these 10 cases were disappointing. Only three patients are known to have achieved complete relief of their symptoms, while four patients benefited little if any, and one patient died after operation.

INTERNATIONAL SOCIETY OF SURGERY

The following abstracts of papers are taken from the program of the International Society of Surgery, Visit to Lyon of the American College of Surgeons, Lyon, France, May 24-26, 1954. The members of the organizing committee were Prof. René Leriche, Chairman; Prof. Paul Santy; Prof. Pierre Wertheimer, and Prof Pierre Mallet-Guy, Secretary.

G. ALLÈGRE: *Diagnosis and Treatment of Traumatic Subdural Hematoma.*

Since the introduction of this lesion into pathology, with Virchow's report, a great number of studies have been stimulated by his methods of diagnosis and treatment. Kronlein and J.-L. Petit demonstrated the importance of the free interval between the time of the trauma and the appearance of symptoms. Henschen outlined the surgical treatment, which was later systematized, as was the diagnosis, by Putnam and Cushing. The introduction of cerebral angiography and electroencephalography permitted the replacement, in most cases, of the injection of air, always a risk in wounded persons, and introduced precision into the diagnosis and prognosis of this lesion.

The 69 cases which are the object of this note were observed during the last four years at the Neurosurgical Center of the Edouard-Herriot hospital. They were selected from a series of more than 300 cases. Analysis of this series of wounded persons has enabled us to rediscover previously known ideas, notably those concerning the violence of the initial cranial trauma (65% of cases). The free interval varies between six days and many years. The shorter the interval the graver the prognosis, for of five wounded persons with a free interval of 5 to 10 days all are dead.

The "free interval" phase is terminated by the sudden appearance, one time out of two, of symptoms which rapidly become worse; in the other half of the cases the development of symptoms is progressive.

The presence or absence of cranial fracture is not significant.

Clinically, the wounded may be classed in four categories:

1. Those with headache (38 cases). In four cases vomiting was associated with headache. This is a

classic symptom, which indicates that other examinations should be made.

2. Those with signs of progressive pyramidal involvement. Sometimes these signs disappear, then reappear. This manner of development, of appearing and then disappearing, seems rather characteristic of a probable subdural hematoma. The value of these pyramidal signs and of the Babinski reflex is found to be unreliable about one time in five.

3. The third clinical type is represented by the wounded who are in coma at the time of hospitalization (25 cases). Nineteen had no sign of localization; 6 had motor deficiency or an ocular paralysis. Here, again, mydriasis was found to be an unreliable sign in half the cases.

4. The last clinical type is represented by those with epileptic attacks (eight cases). These were of the grand mal type.

In the light of this clinical information, the classic schema of hematoma is restated thus:

(1) the injury

(2) the free interval

(3) symptoms of headache; pyramidal signs

(4) phase complicated by coma.

Under these conditions the clinical diagnosis is not correct in more than 40% of cases.

Can this poor percentage be improved with the aid of electroencephalography? Analysis of the records of 54 wounded persons permits us to place them in two groups (1) those with diffuse anomalies in the two hemispheres (14 cases) and (2) those with anomalies in one hemisphere or localized in the frontotemporal region (40 cases).

Thirteen records showed low voltage; 40, delta waves; 25, theta waves, and 9, pointed waves. This series does not permit the establishment of a pattern characteristic of subdural hematoma, as others have described. We believe that electrography provides more indications of prognosis than of diagnosis. Of 10 patients with points or pointed waves and a very slow rhythm, with notched waves, 4 of 6 patients are dead; all the others are cured. The relative worth of electrography for diagnosis is replaced by its prognostic interest.

In contrast to this relative information, the positive value of cerebral angiography may be cited. This demonstrates the existence of the hematoma and indicates precisely the place to operate. This examination may be done on both sides without a disproportionate risk to the patient.

These signs allow prediction of the volume of the hematoma by showing the presence or absence of penetration under the falx of the brain, information of great value for prognosis.

At the present time, with the aid of these two supplementary examinations, the percentage of exact preoperative diagnosis is raised to 92%.

The operative findings are too well known to the reader for us to dwell upon the appearance of the hematoma or its volume. On the contrary, the appearance of the flattened, reddened, and abnormally depressible cortex is an element of bad prognosis; we may presume difficulties in cerebral reexpansion.

In these cases, injection of isotonic sodium chloride solution into the ventricle or the meningeal medullary space is a good practice. Four patients were benefited by this procedure.

Evacuation of the subdural hematoma through a small craniotomy is a simple procedure, preferable to an extensive intervention, in itself a danger; of 69 wounded persons, we have lost 11 who were operated upon.

The postoperative mortality is heavy for subjects operated upon during coma or those having very large hematomas (9 of 11 patients died).

J. AVET: *Cranial Traumatisms in Children.*

One hundred eighty-two instances of cranial traumatisms in children under 14 years have been selected from the files of Professor Wertheimer. There were 46 girls and 136 boys (75%). Rare during the first year (three cases), the incidence of injuries increases regularly with age. There were 60 falls, 53 street accidents, 27 bicycle accidents, 12 direct injuries to the head, and 2 miscellaneous injuries. There were 89 fractures of the vault of the skull (79%) and 19 of the base (17%).

The lesions of the vault are grouped as follows:

1. Separation of sutures, 39 cases (35%); 31 were simple, 8, separation-fractures. These were frequent in the first 5 years (51%), rarer in later years (30% from 5 to 10 years, 19% from 10 to 14 years).

2. Fractures, 47 cases (42% of lesions of bone); these are little different in their characteristics from those of adults. Ramified or comminuted fractures were rare.

3. Injuries causing depressions or ridges, 19 cases (17% of bone lesions); 9 were parietal; 7, frontal; 2, temporal, and 1, occipital.

4. Plastic deformations, either "Celluloid ball" or "green wood" injuries, are rarer than the classic works state.

Fractures of the base are much rarer than in adults. There were 19 cases (17% of bone lesions), of which 13 occurred after the age of 10. Ten fractures affected the middle table; 4, the anterior table; 3, both tables, and 2, the posterior table. Seven, or 43%, were from fractures of the vault which radiated to the base.

As in adults, it is impossible to correlate the anatomical lesions with the clinical types.

A. One hundred twenty-nine (70%) of the wounded presented no lasting clinical sign except

for the initial loss of consciousness; 20 manifested transitory Babinski reflex, mydriasis, and abolition of reflexes; 109 were normal from the return of consciousness.

B. Thirteen cases (7%) presented a motor deficiency of the limbs (hemiplegia in 10, monoplegia in 2, quadriparesis in 1), immediate in 11 cases (cerebral contusion), secondary in 2 (1 with subdural hematoma, 1 with cerebral edema on the second day). Ten patients recovered completely (71%).

C. Paralyses of cranial nerves. Four patients had immediate paralysis of the seventh nerve (two central, two peripheral); seven had secondary paralysis of the seventh nerve; three, of the sixth nerve, and one, of the third nerve; all recovered.

D. Thirteen patients had convulsions.

E. There were five meningeal hemorrhages, three extradural hematomas in older children (9, 11, and 14 years old) who were cured by operation, one subdural hematoma, and three cases of meningitis.

Although, among the elements of prognosis, the loss of consciousness and the pulse are without great value, the rising of arterial tension is a very grave prognostic sign. Mydriasis does not seem to be very serious, in contrast with myosis and nystagmus.

The electroencephalogram, as in adults, does not always give certain proof of cerebral lesions; diffuse alterations are of less value than focal signs.

The mortality is 5%.

Sequels occurred in 22 patients (12%). There were four cases of epilepsy, four of paralysis (one hemiplegia, two monoplegias, one paresis), one case of deafness, one of strabismus, one of hemanopsia, two of facial paralysis, six of headache, one of trouble with speech, and two of character change.

J. Lecuire: *Coma in Recent Closed Craniocerebral Injuries.*

If loss of consciousness indicates a blow to the brain in a case of cranial injury, profound and lasting alterations of consciousness give evidence of lesions localized in the basal region of the brain, the floor of the third ventricle, the hypothalamus, and the mesencephalic portion of the cerebral trunk.

Experimental studies have established the importance, in the waking state, of a system the seat of which corresponds to the reticular substance of the mesencephalon. A localized destruction of these regions provokes prolonged coma in animals. Electroencephalographic studies show, on the other hand, that these lesions reverberate with cortical and subcortical activity. My observations have been made on more than 100 patients in whom coma was prolonged, lasting from 4 to 39 days. More than three quarters of these patients were alive three months after the accident.

Detailed clinical and neurologic examinations, special investigations (encephalography with gas, cerebral angiography, exploratory openings with a trephine), and repeated electroencephalographic studies have attempted to locate precisely the mesencephalic damage. Anatomopathologic verification in a certain number of wounded persons who have died has confirmed the frequency of destructive lesions of the central part of the cerebral trunk where the reticular substance is situated.

These clinical, electrographic, and anatomopathologic data agree with experimental findings.

It is difficult to state precisely the respective frequencies of primary lesions of the cerebral trunk which have repercussion upon the cerebral hemispheres and mesencephalic lesions secondary to those of the hemispheres. The associated injury of the neurovegetative centers of the cerebral trunk and of the base of the brain plays a considerable role in the prognosis of life in these prolonged traumatic comas.

G. Arnulf: *Surgical Treatment of Angina Pectoris by Resection of the Preaortic Plexus.*

The attack of angina pectoris is provoked by either spasm or thrombosis of the coronary arteries.

The ideal objective of effective surgical treatment is not only to suppress the pathways of pain but also to create coronary vasodilatation.

The coronary arteries are innervated by sympathetic and vagal fibers. All these fibers converge in front of the arch of the aorta, where they form the preaortic plexus. These fibers rejoin the coronaries, passing in front of and behind the pulmonary artery or often in the interstice which separates the base of the aorta from the pulmonary artery. The majority of these filaments come chiefly from the left cervical sympathetic and the left vagus nerves. Some come from the right sympathetic, reaching the coronaries by passing in front of the base of the aorta. Finally, some sympathetic or vagal fibers arise from recurrent fibers passing under the aorta to rejoin the preaortic plexus. The preaortic plexus, then, is formed by a series of five or six preaortic fibers and some fibers from beneath the aorta.

Note particularly that all these fibers can be resected in one operation which gives access to the arch of the aorta by the anterior mediastinal route. Thus, anatomically, the extended resection of the preaortic plexus results in a complete denervation of the right and left coronaries.

An experiment has been made on dogs, whose anatomical arrangement is very similar to that of man so far as the preaortic plexus and the distribution of its branches to the coronaries are concerned.

1. Section of the preaortic plexus diminishes coronary and pericardiac sensibility.

2. Resection of the preaortic plexus results in coronary vasodilatation.

This essential fact is proved by the measurement of differences by the thermostromuhr of Reihn and, chiefly, by the method of the graduated index. This, graduated in 0.5-mm. intervals, is placed under a branch of the coronary artery. It shows vasodilatation in a significant way; after section, this attains an average of 28%.

In man the preaortic plexus can be affected by

1. Infiltration. This technique is very simple and without danger. Of thousands of infiltrations in practice, I know of no accident. To obtain a lasting effect, it is necessary to make a series of infiltrations spaced over some days. Usually, 12 are sufficient. New series can be done later.

2. Resection by the extrapleural anterior mediastinal route. Twelve centimeters of the second left rib is resected. After ligature of the internal mammary vessels, the left and right pleura are separated. The left pulmonary hilus is exposed at the deeper level, and the left vagus nerve is identified, an essential identification. All the fibers situated within the vagus as far as the anterior face of the ascending aorta are resected. To make a more complete resection it is necessary to section the mass of fibers which are found beneath the aorta and which represent the posterior anastomoses.

The method should be restricted to all serious cases of angina in which the medical treatment does not give relief or in which infiltrations give transient relief, whether or not there are signs of coronary obliteration.

The results, in 22 cases in which operation was done, show no operative mortality and complete disappearance of the pain in 80% of cases. The long-term results (many years in most cases) include three deaths; two were from noncardiac causes, and the cause of one is unknown.

J. Sautot: *Heterogenous Arterial Grafts.*

I have experimentally produced heterogenous arterial grafts with constant success (carotid artery of calf grafted onto abdominal aorta of dog).

Sudden refrigeration to a very low temperature (—72 C) and storage in a medium containing 10-(2-dimethylamino-1-propyl)phenothiazine (promethazine; Phenergan) and heparin appear to be the principle elements of this success.

Arteries preserved in this manner present an absolutely integral histologic structure for six months.

Clinical, arteriographic, and macroscopic data show that permeability is preserved and that there is neither aneurysmal dilatation nor calcification of the wall of the graft, even after 18 months.

Histologic examinations, on the contrary, each time show degeneration of the muscular fibers of the media.

It is not a case, then, of "arterial grafts" in the true sense of the word but of well-preserved tissue prostheses.

I have made 14 heterogenous arterial grafts in man, without incident or accident, either of necrosis or of sudden thrombosis, and with encouraging success.

P. Wertheimer and J. Descotes: *Long-Term Results of Retrogasserian Neurotomy.*

Of 441 cases of true facial neuralgia, observed until December, 1952, 313 were treated surgically. Among these interventions the operation of Spiller-Frazier occupies a preponderant place, since it was used in 299 cases of primary facial neuralgia. If one adds to this figure 5 contralateral radicotomies for bilateral neuralgia and 14 reinterventions for relapse, one obtains a total of 318 operations performed by the temporal route.

These 318 operations by the temporal route were often performed on older patients (58% of the patients were older than 60 years).

The risks are slight. There were five deaths, giving a mortality of 1.6%; the frequency of ocular complications was of the order of 8%; those of facial paralysis, about 3%. The immediate results were excellent; 98% had good results; only five failures were observed. On the points of benignity of the intervention and excellence of the immediate results all the statistics are in agreement.

But it is much more interesting to consider the long-term future of these patients who are operated upon, as it is shown by the analysis of 195 long-term results (195 followed for more than one year, of which 126 were followed for more than three years).

The long-term results are as follows: 1. Only 73% of patients are satisfied with the result of the intervention (in contrast with 98% satisfied with the immediate results). Further, not all of these satisfactory results are of the same quality—47% of the results are perfect; 26% of the cured patients have slight paresthesias. 2. In 27% of cases the results have become poor or frankly bad after a period of complete relief which was believed to be a definitive cure (this delay is more than three years for more than half of the patients). This group of nonsatisfactory results includes (*a*) 3% with true painful paresthesias, (*b*) 14% with partial relapse (patients suffer attacks, but in a fashion less intense and less frequent than before surgery), (*c*) 10% with true relapse; the intensity and the frequency in general necessitate reoperation.

In almost half the cases the reoperations have shown the completeness of the radicotomy and have resulted in failure. The new tentative therapies fail equally—sleep cure, lobotomy, and peripheral neurotomy.

These long-term relapses show that one ought not to be satisfied with immediate favorable results in studying the results of surgery in trigeminal neuralgia, but one ought not to forget that these

statistics show, beside these 30 long-term relapses, 100 confirmed cures after more than three years.

The Spiller-Frazier operation remains, in the case of essential facial neuralgia, the operation most benign and most sure in its results.

L. MASSUY: *Results of Surgical Treatment of Refractory Sciatica.*

In the service of Professor Wertheimer, 421 patients were operated upon with the diagnosis of "lumbosciatica of discal origin," the diagnosis being established clinically and by simple roentgenography, without any myelography. Operation showed in 266 cases (63%) a discal lesion (hernia, protrusion, or discomalacia) and in 31 cases diverse lesions (ostiophytes, hypertrophy of the yellow ligaments, arachnoiditis). In 118 cases exploration was negative; 6 patients had only a disinsertion of the sacrolumbar masses without intraspinal exploration. Fourteen patients were operated upon again; in 3 cases a hernia was found which had not been found at the first operation; in 2 cases there was a recurrence of discal hernia; 1 patient had arachnoiditis which resulted from the operation; another had a subdural hematoma, also following operation; in 7 others no lesions were found other than those which had been found the first time. Finally, in 26 cases, the first or second intervention had been completed by a bone graft; these patients presented congenital or acquired vertebral malformations.

Among these 421 patients, only 337 were seen again after at least three months of postoperative evolution. The results are analyzed as follows:

There were 159 (47.2%) complete cures; 112 (33.2%) satisfactory results, that is, persistence of some sciatic pain with fatigability, amyotrophy, and vertebral stiffness but without important difficulty; 66 (19.6%) failures. The best results have been obtained by ablation of the hernia with or without radicotomy. The grafts gave the largest percentage of failures (36%). The sequelar disorders most often found are persistent lumbalgia, reappearances of sciatic pain, and, finally, disorders of motor or sphincteral insufficiency.

P. MALLET-GUY: *Surgical Treatment of Chronic Relapsing Pancreatitis.*

The clinical history of chronic relapsing pancreatitis is made up of attacks of gastric pain radiating to the left, repeated irregularly, of unequal intensity, accompanied by more or less masked peritoneal signs, and preceded or followed by the syndrome of chronic painful dyspepsia. It is also characterized by the infrequency of signs of glandular deficiency, either exocrine or endocrine.

My first personal cases were collected in 1934, making possible in 1938 the description of the syndrome, which is similar to that which Comfort, Gambill, and Baggenstoss reported in 1946.

Surgical intervention for chronic relapsing pancreatitis must include (*a*) complete exploration of the pancreas, head, body, and tail, and, if necessary, a biopsy and (*b*) complete exploration of the biliary tree, comprising preoperative manometric and roentgenographic studies, to show precisely if there are biliary stones, obstruction of the common duct, or dystonia of the sphincter of Oddi.

Compression of the common duct in chronic pancreatitis may be treated by anastomosis between the biliary and digestive tracts—cholecystogastrostomy, cysticoduodenostomy, or choledocoduodenostomy—palliative and not curative treatment of chronic recurring pancreatitis.

Dystony of the sphincter of Oddi may be the initial cause of chronic pancreatitis, by the mechanism of reflux of bile in the canal of Wirsung; rarely, it will be hypertonicity (only indication for vagotomy or sphincterotomy); oftener, it will be hypertonicity of the sphincter of Oddi (indication for right splanchnicectomy).

In less clear-cut cases, the choice of operation is guided only by the concept of an evolution schema of chronic relapsing pancreatitis: (1) at the beginning, a primary episode of pancreatic autodigestion; (2) if the necrotic lesions are very intense and very extensive, a pseudocyst of the pancreas develops; (3) if the lesions are less intense, sclerosis follows the edema and patches of necrosis; (4) these foci of sclerosis are the starting points of abnormal vasomotor reflexes which induce new acute episodes in other parts of the gland and thus establish a disposition to chain reactions.

This hypothesis is supported by the nerve lesions often encountered in chronic relapsing pancreatitis: edema or thickening of the nerve sheath, perineural islands of inflammation, neuroma formation and hypergenesis, and degenerative lesions of the protoplasm and nuclei of the ganglion cells.

Such a reflexive cycle of chronic relapsing pancreatitis can be interrupted by suppression of the focus of the sclerosis and the nerve lesions, starting point of the reflexes—left pancreatectomy.

The first case dates from September, 1934. The development had been particularly severe, having already necessitated two emergency operations. Left pancreatectomy resulted in complete cure, confirmed after 18 years.

As of February, 1944, there have been 14 cases of left pancreatectomy for chronic relapsing pancreatitis. There were 2 postoperative deaths (shock, diencephalic complication); 10 excellent results (4 after 18, 18, 15, and 14 years, respectively; 3 after 4 years, and 3 after from 32 to 40 months); 2 early recurrences in the head of the pancreas (the third and fourth cases, observed 17 years ago, which show the necessity of strict limitation of the lesions to the left half of the pancreas).

The reflexive cycle of chronic relapsing pancreatitis can be interrupted by section of the reflex arc, left splanchnicectomy, the justifiability of which is confirmed by experimentation.

Electric stimulation of the left splanchnic nerve in the guinea pig induces, successively, pancreatic vasodilatation, edema, and hemorrhage.

Only excitation of the whole left splanchnic nerve and of its peripheral end produces acute pancreatitis. Excitation of the central end of the left splanchnic nerve, of the right splanchnic nerve, or of the vagus nerves is ineffectual.

Secretory activity and anaphylactic shock are aggravating factors.

Two series of observations permit exact statement of the value and the conditions for use of sympathetic operations in chronic relapsing pancreatitis: (*a*) left subdiaphragmatic splanchnicectomy, 52 personal cases from June, 1942, to November, 1951, and (*b*) splanchnicectomy plus unilateral or bilateral thoracolumbar sympathectomy, 29 cases published, during the same period, by various U. S. surgeons after Geza de Takats (1947).

In my personal series, splanchnicectomy was always preceded by an exploratory laparotomy including manometric and roentgenographic examination of the biliary tract. It was done by the subperitoneal lumbar approach and was completed, in nine cases, by resection of the left semilunar ganglion. The results were as follows:

1 death (mortality 1.9%)
9 cases followed less than a year (7 perfect results, 2 improvements)
42 cases followed from one to nine years

Failure (secondary pancreatectomy)	1 case
Improvement	5 cases
Good result (no pain, some functional trouble)	9 cases
Perfect results	26 cases
Immediate	20 cases
Secondary	6 cases
Total good or perfect results	35 (83.3%)

The immediate perfect results were characterized by immediate disappearance of pain and functional troubles (diarrhea), cessation of diet restrictions, and rapid weight gain of 10 to 20 kg. (10 patients had already undergone, without success, one or two biliary operations, and in 3 cases there were pancreatic calcifications).

More rarely, during six months, some symptoms may persist and new attacks may occur, "miniature" attacks which are milder each time. Then cure becomes complete, with progressive regain of weight (19 kg. in one case)—the secondarily perfect result.

The result is called good when, in spite of disappearance of attacks of pain, there is persistence of some digestive troubles, necessitating diet, and, rarely, some very mild attacks of pain.

The quality of these results permits one to affirm that left splanchnicectomy is not only a symptomatic operation against pain but also a curative operation for relapsing pancreatitis. It effectively interrupts the evolutive cycle of the disease.

I further state that it is efficacious in pancreatitis with calcifications but not in lithiasis of the duct of Wirsung, that it is not demonstrated that radical sympathectomy gives a better percentage of cure, and that bilateral intervention is useless.

J. Feroldi: *Upper Pancreatectomy in the Treatment of Necrotic Pseudocysts of the Pancreas.*

The treatment of necrotic pseudocysts of the pancreas is classically performed by formation of a shunt, either external, often followed by a fistula, making a secondary anastomosis necessary, or internal in the first place (anastomosis between the cyst and the intestine), the long-term results of which are few and to which objections based on principle may be opposed: the anastomosis creates a large diverticulum, of which the drainage may be hazardous.

Since 1942, P. Mallet-Guy has substituted for the shunt an operation of excision, excision not of the cyst, which has no true wall, but of the upper segment of the pancreas (seven personal cases).

A first argument in favor of upper pancreatectomy is that, in each case, examination of the cavity shows an interruption of Wirsung's duct, largely open to its interior. This duct has become necrosed in the course of the initial attack of pancreatic autodigestion, and that destruction is the cause of the evolution of the pseudocyst, which collects the secretions of the upper pancreatic segment.

The resection of this segment of the gland suppresses the cause of the pseudocyst and assures the prevention of a fistula, for a reflux from the inferior end of the canal is not to be feared, even if it has not been possible to tie it.

The first case observed was of a patient first operated on for external drainage. A pancreatic fistula persisted, in which roentgenologic examination revealed, in showing the lower end of Wirsung's duct, interruption of the canal in the pseudocystic cavity. Removal of the upper part of the pancreas dried up the fistula in 10 days and gave a perfect cure, confirmed after five years.

In the second patient upper pancreatectomy promptly put an end to a long history of chronic relapsing pancreatitis, already complicated by suppuration, temporary fistula, and, finally, a pseudocyst; in the operative specimen the orifice of ulceration of Wirsung's duct could be clearly seen.

The same lesion was found in a third case. In a fourth case, it was more complex, with double ulceration. A fifth observation had the value of an experimental document: a fistula, which appeared after pancreatectomy and was maintained by the left end of the pancreas, which had been left in place, was dried up by the secondary excision of that fragment.

A second argument in favor of left pancreatectomy in the treatment of pancreatic pseudocysts is that, in four of the cases, preceding the attack of pancreatic necrosis, there was a more or less old syndrome of chronic relapsing pancreatitis and that, each time, examination of the excised pieces effectively showed very accentuated histologic lesions of chronic pancreatitis.

Thus, pancreatectomy assures at best, after cure of the pseudocyst, prevention of acute episodes of relapse.

A third argument in favor of pancreatectomy is again histologic: in two other cases (that is, two of seven operated upon) malignant neoplastic lesions were discovered, most unexpectedly, in the excised specimen, although the clinical history and the operative findings led to the diagnosis of common necrotic pseudocyst of the pancreas.

One of the two patients had a rapid relapse; the other is still well six years and six months after operation, even though, histologically, a lymph node had already been invaded.

It may therefore be assumed that

1. Acute autodigestion of the pancreas may lead to necrosis of Wirsung's duct. The secretions of the upper pancreatic segment, thus turned toward the necrotic focus, augment the encysted, pseudocystic collection.

2. The upper glandular segment is the seat of marked inflammatory histologic lesions of the type of chronic relapsing pancreatitis.

3. It may also be the seat of an epithelioma.

These three facts justify, in postnecrotic pseudocysts of the pancreas, left pancreatectomy, of the upper part, in preference to anastomosis operations.

R. Feit: *Technical Conditions of Manometric and Roentgenologic Study of the Biliary Tract During Operation.*

There is a high percentage of relapses in surgery of the biliary tract. These relapses are not only due to technical faults but also (1), in the course of cholecystectomy, to not recognizing an anatomical or functional obstacle in the principal bile duct; (2) after choledocotomy, to the impossibility of affirming, even by catheterization, that the principal duct is clear; (3) in gall bladders with stasis but without stones, to the routine use of cholecystectomy.

Analysis of the statistics of reoperations for the syndrome of relapse after cholecystectomy shows that, in the greater number of cases, the factor causing relapse existed well before the first intervention. No method of examination, even the most recent roentgenographic techniques (iodopanoic acid [Telepaque]; sodium iodipamide [Biligrafin]) can consistently prevent errors.

In 1932, Mirizzi proposed, to prevent such relapses, cholangiography during operation.

But it is often difficult to interpret the negatives without fluoroscopy, practically impossible in the operating room. A manometric reading may be substituted for it; the curves of biliary pressure are equivalent to fluoroscopic visualization (P. Mallet-Guy, 1942).

The technique comprises two successive and independent steps:

(1) manometric recording of a test of hyperpressure by injection of isotonic and isothermic sodium chloride solution into the gall bladder or the common duct.

(2) cholangiography, including, after injection of a contrast medium, taking two or three films at three-minute intervals.

The two methods check each other, and their results should be easy to read and to interpret.

One has thus a physiopathologic biopsy which will be compared with the clinical findings and those of the laboratory and of the exploratory operation to permit the surgeon to make a diagnosis.

Used routinely, the manometric and roentgenographic data make possible the discovery of unsuspected facts, even in cases which are apparently the simplest.

Prof. P. Mallet-Guy's series of cases had increased, by Feb. 1, 1954, to 2,100 interventions and has been complemented since 1947 by more than 300 experiments on dogs.

These experimental researches permit the following statements:

1. The tonus of the biliary tract is not disturbed by injection of an isotonic and isothermic saline solution.

2. Local anesthesia is not necessary. General anesthesia with nitrous oxide-ether-oxygen does not disturb the functional state of the biliary tract in the absence of all premedication, complementary medication, and curarization.

3. Water-soluble contrast mediums produce various perturbations of biliary pressure; methane-iodine by-products provoke hypertension by spasm of the bile ducts, whereas the iodo-oxypyridines (iodopyracet [Diodrast]) disturb, either by raising or by lowering, the biliary pressure.

4. On the other hand, liposoluble iodine products do not produce troubles of tonicity.

In all cholecystectomy for lithiasis without common duct signs, after negative exploration, the cystic duct is freed and sectioned on half of its circumference.

A bent cannula is introduced and engaged in the common duct.

The common duct pressure is normally 10 to 12 cm. of water; above 15 cm. it is hyperpressure; below 7 cm., hypopressure.

In reoperations for relapse the cystic stump will also be located, and, without further dissection, the cannula is placed through it into the common duct.

Whenever cholecystectomy is not justified, the gall bladder is punctured (normal pressure, 18 to 20 cm. of water). Thus, in gall bladders with stasis, the pathologic physiology can be analyzed by extemporaneous manometric and roentgenologic study.

M. Gangolphe: *Manometric and Roentgenographic Recording During Operation in Surgery for Lithiasis of the Common Duct.*

Manometric and roentgenographic control during operation allows (1) discovery of unsuspected stones of the common duct; (2) verification, after choledocotomy and extraction of the stones, that a forgotten stone does not still remain; (3) determination of the functional state of the sphincter of Oddi and the possibility of compression of the common duct by chronic pancreatitis, in this manner aiding in the choice of the mode of operation.

These figures are based on 100 operations for common-duct stones, from June 14, 1950, to July 22, 1952 (P. Mallet-Guy), out of a total of 530 biliary operations:

Chronic jaundice, angiocholitis	32 cases
Acute attacks of pain with chills, high temperature, transient jaundice (syndrome of Eugene Villard, Lyon, 1909)	38 cases
No common duct symptom	30 cases

Of the 30 cases out of 100 in which no clinical signs of common duct stones were apparent, 9 times they were discovered by palpation of the common duct, after disengaging the duodenum and pancreas; 8 times they were suspected by the dilatation of the common duct but were not perceptible by touch; 13 times they could not even be suspected but were discovered by manometric and roentgenographic control during operation.

These stones, clinically inapparent in the common duct, were found in 10% of cases of common lithiasis of the gall bladder. This is the proportion of common duct stones found in cases of relapse after cholecystectomy, stones preexistent and not consecutive to cholecystectomy.

After choledocotomy and extraction of stones, when every obstruction, according to catheterization, seems to have disappeared, manometric and roentgenographic control can show that one or several stones or fragments of stones remain in 4% of cases.

Thus will be avoided a biliary fistula or a recurrence of jaundice after choledocotomy.

The surgeon is led to choose between (*a*) external biliary drainage, (*b*) primary suture of the common duct, and (*c*) choledocoduodenostomy.

External drainage after choledocotomy has often been recommended and each time has been abandoned because of fear of the risk of choleperitonitis due to rupture of the suture.

The "ideal choledocotomy" formally condemned in France and in England since 1897, retains, in spite of Kehr, strong supporters in Germany and in Austria.

Choledocorrhaphy can be reinstated in lithiasis of the common duct by the use of manometric and roentgenologic control during operation (from October, 1947, to June, 1950, 25 cases).

Required conditions are (*a*) absolute freedom of the common duct, (*b*) aseptic bile, and (*c*) strength of the common duct wall.

From June, 1950, to July, 1952, there were 19 cases out of 100. "Ideal choledocotomy" has been used either in benign cases (10 cases, no mortality) or in serious cases, to avert, in a delicate patient, the risk of biliary drainage (9 cases, 1 death on the eighth day of coma).

Choledocoduodenostomy is easily done on a very dilated common duct, with thick and resistant walls. Infection is not a contraindication.

Manometric and roentgenologic control affirms the absence of stones and can show disease of the sphincter of Oddi or compression due to chronic pancreatitis.

Thus, choledocoduodenostomy sometimes averts a biliary fistula and always prevents the great loss of bile which external drainage of the dilated bile ducts entails. It is therefore particularly indicated for delicate patients.

It has been performed 54 times in the 100 patients, of which 15 were reoperations for recurrences. Twenty-one patients were over 60 years; 15, over 65, and 10, over 70. Of 32 cases of small or moderate risk there were 2 deaths; in 22 cases of bad risk there were 3 deaths.

J. Descotes: *Nerve Operations in Treatment of Biliary Dystonias.*

Dystonias of the bile ducts are frequently encountered in surgical practice. They clarify the problem of "stasis of the gall bladder" and explain the frequent failure of medical treatment and the bad effects of nonjustified cholecystectomy. But above all, dystonias of the sphincter of Oddi are important factors in relapse after cholecystectomy. Therefore they should be searched for in the course of each operation for gall bladder lithiasis.

In their treatment, a preponderant place has been given since 1941 to nerve operations to correct the tone of the biliary tract (P. Mallet-Guy).

It has been shown experimentally that right splanchnicectomy decreases the tonus of the biliary tract and the sphincter of Oddi (P. Mallet-Guy and R. Guillet, 1941).

Precise diagnosis of biliary dystony can only be made during operation, by manometric and roentgenologic studies.

Hypotonicity of the biliary tract is manifested by a syndrome of pain, sometimes fever or even jaundice, severe dyspepsia, stubborn headaches, a marked general attack, and frequent failure of medical treatment.

Right splanchnicectomy, technically very simple, is sufficient to assure the treatment of syndromes of biliary hypotonicity; there is no nerve regeneration for a long time (extensive sympathectomy is not necessary), and objective results are given by later postoperative cholecystography and repeated manometric and roentgenographic studies.

One hundred fifty-one right splanchnicectomies performed in this way from 1941 to 1950 gave an operative mortality of four, or 2.6%; three pulmonary complications, and one gastroduodenal dilatation (resection of the right semilunar ganglion).

One hundred thirty-five patients were followed; failures were 8%; relapses, 4.5%; improvements, 5%; good results, 30.5%; functionally perfect results, 52%.

It is possible, symmetrically, to affect conditions of biliary hypotonicity by vagotomy.

Doubtless, in cystic disease cholecystectomy ought to be done in all cases, but in disease of the sphincter of Oddi vagotomy is a rival to transduodenal sphincterotomy. It is to be preferred if there are no sclerotic lesions of the sphincter.

The differential diagnosis between labile hypertonia (vagotomy) and fixed hypertonia (sphincterotomy) of the sphincter of Oddi is made by infiltration of the vagus nerves with procaine hydrochloride (Novocain) during operation, at the cardia or along the lesser curvature of the stomach.

Vagotomy at the cardia creates a risk of postoperative visceral dilatation, and perhaps, logically, it seems excessive to suppress vagal innervation of the abdominal digestive tract in order to treat a very localized state of muscular hypertonia. Thus, one may debate between right, left, or bilateral vagotomy; high vagotomy, at the cardia, or low, cutting all the branches at the middle part of the lesser curvature.

The clinical results, which well illustrate in cases of high vagotomy the risk of gastrointestinal dilatation, seem to demonstrate, on the other hand, the effectiveness of low vagotomy, which does not have this consequence.

Experimental study of the effects of vagotomy on the tonus of the bile ducts shows (1) that vagotomy at the cardia, always effective if bilateral, is almost but not quite always effective if limited to the left side, whereas the results of right vagotomy are very irregular; (2) that the effects of low bilateral vagotomy are clear and regular, a little less intense than those of bilateral vagotomy at the cardia but not accompanied by gastric troubles.

Low vagotomy thus appears in all respects to be the operation of choice in labile hypertonicity of the sphincter of Oddi.

L. Eichholz: *Indications and Results of Periarterial Neurectomy of the Hepatic Artery in the Treatment of Hepatitis.*

Possible indications for operation in jaundice in hepatitis are as follows:

A. Diagnostic errors. Cholangitis syndrome; painful attacks with fever, followed by jaundice, leading to the diagnosis of lithiasis of the common duct, which condition does not exist, the jaundice being caused by intrahepatic lesions, "cholangitis." Chronic jaundice, characterized by humoral tests of obstructions; supposed diagnosis, compression of the common duct, but the cause is an obstruction of the minor intrahepatic channel, mesenchymatous hepatitis, or hepatosis with biliary stasis.

B. Jaundice getting worse, with failure of medical treatment, an indication for operation as the last chance.

Historically, Umber, in 1919, operating for jaundice in hepatitis, obtained a cure after simple laparotomy. Then, in 1922, Tietze attempted a biliary shunt, an illogical operation in the absence of biliary lesions, but one which gave some success.

However, the results of such interventions are very inconstant. Before 1947, the personal records of Prof. Mallet-Guy gave the following results:

	Total	Death	Failure	Improvement	Cure
Simple laparotomy	8	2	3	2	1 (12.5%)
Biliary shunt	7	1	2	2	2 (28.6%)

The partial success of the biliary shunt operation may be explained by the existence of a dystonia of the sphincter of Oddi. And manometric and roentgenographic recordings during operation should discover hypertonia in certain cases, indication for a sphincterotomy or for vagotomy, or hypotonia, amenable to right splanchnicectomy. This practice has given, in 15 cases, 1 death, 3 failures, 2 improvements, and 9 cures (60%).

If, the diagnosis of jaundice in hepatitis being confirmed by operation, the bile ducts are, on the contrary, anatomically recognized and functionally normal, since 1947, the treatment of hepatic lesions has been by periarterial neurectomy of the hepatic artery (P. Mallet-Guy), the great efficacy of which can be affirmed, after the condition has passed the stage of medical management.

The common hepatic artery is found on the floor of the bursa omentalis, and the operation, usually simple, may become difficult or even impossible owing to the volume of the liver or be slowed by the presence of large lymph nodes in front of the artery.

From January, 1947, to January, 1954, 50 cases of "medical" hepatitis have been treated by periarterial neurectomy of the hepatic artery.

A. Chronic jaundice in hepatitis (20 cases). In each case there was immediate amelioration of the jaundice. There was 1 death on the eighth day, of uremia; 6 cases were followed less than a year, with very favorable results; 14 cases were followed from one to seven years, with 3 failures, 2 incomplete cures, and 9 stable cures.

The three long-term failures of periarterial neurectomy of the hepatic artery for jaundice in hepatitis corresponded to cases of jaundice which had evolved for 22, 7, and 9 months, respectively. Each time, there was temporary amelioration, then relapse developed. It is concluded that after six months of jaundice hepatic lesions are irreversible.

The stable cures corresponded to very diverse lesions—there were four cases of hepatosis, but there were also cirrhosis, with jaundice of five and one-half months' and of two months' duration, cured after one year; mesenchymatous hepatitis, with jaundice of three months' duration, cured after four years, and chronic cholangitis of 10 years' duration, with jaundice of one month's duration, cured after three and one-half years.

The cure was proved by postoperative puncture biopsy.

B. Angiocholitic syndrome without common duct obstruction (21 cases). There were no deaths, 10 recent cures, and 11 long-term results, comprising 2 improvements (some pain) and 9 perfect cures, with return of general health, regain of weight (10, 15, 17 kg.), and cessation of the relapsing syndrome after one to eight years.

C. Discovery of hepatic cirrhosis in the course of an operation for gall bladder syndrome without jaundice or common duct signs. This condition is an excellent indication for periarterial neurectomy of the hepatic artery.

The objectives are two: to alleviate the great risk of immediate decompensation of the cirrhosis and to aid in the cure of the cirrhosis.

The operation was done for this condition in five cases without postoperative incident, and, of four long-term results, there were three lasting cures and one clear improvement.

Finally, in four cases of hepatitis without jaundice (cirrhosis or cholangitis), the indication for operation was admitted with knowledge of the cause after failure of medical treatment; in the four cases there has been a good result for less than a year.

It is still too early to state the exact indications for periarterial neurectomy of the hepatic nerve. But, operating for an apparently mechanical jaundice, a common-duct syndrome, called lithiasis, or for gall bladder stones and finding hepatitis instead, the surgeon may in this manner avert catastrophes and insure cure in the future for unexpected conditions.

M. Guilleminet: *Results of Surgical Treatment of Congenital Dislocations of the Hip.*

In the course of an inquiry on the results of surgical treatment of congenital dislocations of the hip I have already reviewed 50 patients, representing 70 hips operated on. Some results are recent; others date from 11 years ago.

In the present state of my inquiry, the following conclusions are apparent:

Surgical treatment of dislocations of the hip presents incontestable theoretical advantages; its practical results are encouraging only to the extent to which one carefully respects the details of indications and of execution which pertain to each case; many actions apparently justified by anatomical dispositions may be harmful in very young children.

Surgical treatment ought to be reserved for patients in whom orthopedic treatment is impossible or has already failed.

Simple reduction seems to me by far the best method for children less than 5 years old.

The operation of Colonna, together with fair success, has given me a certain number of failures due to progressive stiffness. It seems to me less reliable. Personally, I only use it after 5 or 6 years of age, when simple reduction proves unsuitable; it can then give magnificent results.

P. Stagnara: *Organization of Treatment of Evolutive Types of Scoliosis During the Period of Growth.*

The consultation of vertebral curvature ought to contain, in each case, a very complete set of records, especially x-ray films taken in the functional position and in the position of repose. Calculation of the angles of the neutral vertebrae permits very exact evaluation of the extent of the deviation.

These documents will be reviewed every six months in order to measure the progress or the stability of the spinal curvatures. The most frequent types of nonprogressive curvatures are treated by physical therapy administered by registered physical therapists. Most of the progressing curvatures have no precise etiologies; they are called idiopathic scoliosis. Our experience confirms results of the fundamental work of Ponseti on the prognosis in idiopathic scoliosis. The size of the curvature depends upon its clinical and roentgenographic pattern and the age of the patient at the beginning of the scoliosis.

The various roentgenographic patterns (thoracic, lumbar, thoracolumbar, and combined curves) permit the classification of different types of idiopathic scoliosis into these four groups. The thoracic curvatures are the most extreme, and next in seriousness, the thoracolumbar curves. These curves produce

terrible gibbosities which are not only unesthetic in appearance but which greatly affect the general development of the patient.

When he is adult, he may suffer from respiratory and circulatory disorders that cannot be ameliorated. The early appearance of scoliosis is indeed a grave matter. The curvatures which are noted before the age of eight often reach great size and extreme degree of rotation. And scoliosis in infants poses an even more serious problem.

In any case, the period of prepubertal growth is usually the period of greatest increase of these curvatures; it is a period of bone malleability.

Many forms of scoliosis progress during the period of growth, but they seem to have a specific disease as a constant or occasional cause. We note in particular poliomyelitis, Freidreich's disease, Recklinghausen's neurofibromatosis, and pleuropulmonary suppurations in all symptomatic forms. Pubertal growth seems to aggrevate scoliosis, as though the precarious equilibrium of the organism were destroyed by this growth. It is the same for certain congenital types of scoliosis in which the compensatory curvatures may increase during this same period. However, these curvatures usually become stabilized at the age of 18. The ossification of the iliac apophyses marks the end of the progression of these curves.

When one has studied the differences between the progressive curvation described above and the labile type (particularly during the period of puberty), one notes that most of the traditional treatments for patients with scoliosis are ineffective for the progressive type of the disease. All treatment attempted for such a disease must be followed with extreme precision for fear of incurring the same difficulties. In the Orthopaedic and Children's Surgical Clinic of Professor Guilleminet, a center for the orthopedic treatment of these curvatures has been set up.

The correction of curvatures is achieved by the application of "distraction jackets" modeled after those of Donaldson and Anderson more than those of Risser. We obtain a reduction of 50% of the angle after three weeks of this distraction process. The correction of gibbosities is obtained by the application of Abbott plasters. The correction of the gibbosites often exceeds 50%, and for moderate curvatures the correction or even the temporary inversion of the gibbosities may be obtained.

Maintenance of the reduction of the curve is the major problem in treating cases of scoliosis. Traditionally it is believed that cure can be obtained only by means of surgery, but surgical treatment sometimes fails, especially when it is employed in children before the period of pubertal growth or in cases of lumbar or combined scoliosis, for the lumbar spine is the site of many pseudoarthroses.

Thus we have tried to solve by orthopedic methods those cases which are not amenable to surgical treatment. The fundamental idea was the retention (after reduction) by jackets, associated with re-education by gymnastic methods which could be performed within the jackets. This procedure was suggested by Bohler's experience in treating fractures of the spine. He demonstrated the efficacy of such a method in avoiding secondary collapses that occur with removal of the jacket. We believed this would hold true for the period of bone modification. This treatment has been used for six years at the Orthopaedic Clinic, but it has proved necessary to keep children undergoing this delicate therapy in the hospital for a long time.

As a rule, the results have been good in all cases of progressive scoliosis, both idiopathic and symptomatic, when we have been able to halt the progression just at the pubertal period of growth. Three or four years after the beginning of treatment, these children are in a better condition, judged roentgenologically and clinically, than before the beginning of their illness.

Even in the severest cases, in which the progressive curvation is not entirely halted, this long patient orthopedic treatment had a good effect, when judged in comparison with the results in cases in which spontaneous curvature occurred, without such treatment. Thus we limit this treatment to curvatures occurring in the age period of bone malleability (puberty) and those for which surgical treatment is difficult (lumbar or combined curvatures). We keep a very close check on all our patients, for it is only after the seventeenth year that we can ascertain the final durability of these results.

From the surgical viewpoint we follow the principles enunciated by Hibbs, Risser, and Ferguson, but we have added some modifications that we found useful. These modifications consist of the use of a preoperative correction jacket (with collar) which makes us less apprehensive of the danger of recurrence, and the use of heterogenous osseous transplants which supplies us with the needed material.

Our studies on heteroplastic bone graft, which we have made for five years, demonstrated that the assimilation time required for the assimilation of defrozen cortical calf bone was longer than that required by autoplastic bone grafts; on the other hand, the assimilation of spongious bone grafts of calf is rapid (less than three months). Consequently, we combine the use of compact bone which provides a rigid splint of slow assimilation with the use of spongious bone, which acts as a quickly assimilated cement supported by the rigid armature of the compact bone. Under these conditions, we can expect an early physical reeducation of the patient, beginning in bed, and allowing him to get up with

his corrective jacket after the third, fourth, or fifth month, depending on the case.

Leather braces are worn from the sixth month to the complete assimilation of the bone grafts (18 months to 2 years). During this entire period, daily reeducation is prescribed, for it is known that during the assimilation there are dangerous periods—particularly those of inflexion and the pseudoarthroses that occur between the eleventh and twelfth months.

A. TRILLAT: *Treatment of Closed Fractures of the Tibia and Comminuted Fractures.*

The orthopedic treatment of closed fractures, except in the very young, is often of long duration and not without difficulties such as pseudoarthrosis and stiffness of the knee and ankle joints. I have tried to find a means of open reduction, with osteosynthesis. Kuntscher's intramedullar wire, cerclage, and metallic plates are not appropriate. Finally it was found that the most convenient device is the use of simple screws.

The procedure consists of the following steps:

1. The leg placed in a special traction device designed by me.
2. The incision made by the anterolateral approach.
3. Combined orthopedic and surgical reduction.
4. The screws placed in a perpendicular direction to the axis of the bone, except in exceptional cases.
5. Total closure. Cast.

Twenty-five percent of the patients had tibiotarsal difficulties during the first year; there were no sequelae after the first year.

A. TRILLAT: *Treatment of the Recurrent Patellar Dislocation: Technical Consideration and Films.*

When it is not necessary to correct a genu valgum or to lower a patella, the treatment for dislocation of the knee cap is to transplant it within the proximal end of the tendon.

The chief difficulty is to obtain a satisfactory procedure which will assure consolidation and, at the same time, permit early walking.

The method of Elmslie-Malkin, which is little known, appears to me to be by far the most satisfactory. The motion picture shows this clearly.

A. TRILLAT: *Repair of the Internal Lateral Ligament of the Knee: Technical Considerations and Films.*

In the various publications concerning the technique of repairing the internal collateral ligament, two principal difficulties are always mentioned. The first difficulty resides in the impossibility of using this ligament itself. Actually it is often broken during the initial injury or during the exploratory operations already performed. It constitutes scar tissue that appears usable, but, in fact, the long fibers have been destroyed. Its utilization after removal of fibrous tissue is made impossible by the loss of substance which results from this procedure.

To strain it by lowering the point of attachment at the lower insertion (Mauck's operation) is worthless from the mechanical point of view. This is a procedure that should be rejected.

The second difficulty is to obtain sufficient tension so that the points of attachment fuse solidly enough with the plastic material employed to replace the missing ligament.

After I made studies on cadavers and after reading about the technique of Mac Murray, first published in 1918 and recently advocated by Watson Nones, I concluded that repair operations could not give good results unless the transplanted tendon is not cut. The technique of Mac Murray is accomplished by the simultaneous use of the gracilis muscle and semitendinisus muscle. This will be shown in the films.

The procedure is as follows:

1. Exposing and freeing the lower insertion of the gracilis muscle and the semitendinisus muscle.
2. Making a deep vertical slot in the osseous substance and unsheathing the upper internal lateral ligament.
3. Fixation into the bottom of this slot of the two joined ligaments, without section.

This operation is simple. The tension of the tendons is satisfactory. The tendons utilized can not slip out of the osteofibrous slot through which they pass. The patient can use his leg and his knee early without worrying about breaks or weakening of the sutures.

M. DARGENT: *Parotidectomy with Conservation of the Facial Nerve (Report of Seventy-Four Cases).*

The desirability of doing a parotidectomy, either total or partial, with section of the facial nerve in treating so-called mixed tumors of the parotid gland has been advised since 1943. All the tumors on which operation was done gave a clinically benign impression, and most of them looked like encapsulated lumps of the parenchyma.

The anatomopathologic data assembled suggested the following diagnoses:

30 mixed tumors
8 cylindromas
10 recurrences of mixed tumors
10 encapsulated tumors
5 recurrences of carcinoma with a clinically encapsulated tumor
1 lipboma
1 sarcoma
1 neurinoma
4 inflammatory lesions (nodular tuberculosis)
2 benign cysts
1 adenoma

Fifteen of these were recurrences of a tumor previously encapsulated. With the exception of the lymphoma, the neurinoma, the lipoma, cysts and adenoma, which were really benign, all these tumors must be considered as malignant from the beginning. All the French pathologists consider the so-called mixed tumors as a slow-growing cancer for which the term "changing epithelioma" is used, but we still use the classic term because it indicates a clinically well defined group of tumors that appear benign and are potentially malignant.

In the first group of 50 patients, total parotidectomy was done, and the facial nerve was exposed from its principal root to its farthest branches, thus excising the superficial and deep lobes of the gland. In this way the parotidectomy was as radical as possible, and the patient was protected from the danger of recurrence which frequently followed simple enucleation. Little by little, this procedure has been abandoned as a systematic procedure for the following reasons:

From the anatomopathologic viewpoint, it is clear that the so-called capsule of the mixed tumor is an artifice and that the removal of a tumor by the excision of healthy tissue all around it is necessary, but the presence of malignant foci from the primary lesion in the two lobes described by Delarue in Paris has never been found in any of our surgical cases. Further, removal of the deep lobes, necessitating a minute dissection of the facial nerve and depriving its sheath of its vascularity, is not necessary when the tumor is superficial.

From the practical viewpoint, the facial paralysis that generally disappears after a few days, three weeks, or a few months may remain in certain areas, notably those of the upper and lower lips, or facial spasms may occur. In seven cases this complication was noted. Ephydrosis is also an annoying complication, and it appears that section of the deep portion of the auriculotemporal nerve is responsible for these secretory changes.

Therefore total parotidectomy should be done only for recurrent growths in which the diffusion of malignant foci is apparent even in the skin, for tumors of the deep lobe that develop beneath the trunk of the facial nerve, and for encapsulated tumors identified during the operation.

Block dissection of the neck ganglia associated with parotidectomy is advisable in such a case, but the systematic section of the facial nerve is not necessary. Frozen section or ultropack examination is made during the operation when the tumor has been exposed and the facial nerve brought to light. In the benign cases simple enucleation is done. Removal of the superficial lobe is done for superficial tumors. As the roentgenological pictures of the sialographies are not always characteristic, the exploratory phase of the operation is necessary.

The following results were obtained:

Of 30 patients operated on for mixed tumor, 29 lived from 1 to 10 years without recurrence; 18 of them for 5 years. One was lost to follow-up.

Of eight patients operated on for cylindroma, one died of meningitis the first year; the other seven have lived without a recurrence, four of them for five years.

Of 10 patients on whom operation was performed for mixed tumor, 6 are living without recurrence; 3 are living with recurrences in the cutaneous scar. One died early from malignant growth in the ganglia of the neck. The primary lesion was a mixed tumor. The ganglionic metastases were of the spinal cellular type.

Of 10 patients operated on for encapsulated cancer, 6 are living after from two to eight years; 4 apparently had ganglionic involvement of the neck. Three died of pulmonary metastases associated with malignant cervical growths in two instances.

Of the five patients operated for recurrent nodular tumor, four are living without recurrence from one to seven years after operation.

There was nothing of note in the other cases.

M. DARGENT: *Resection of the Upper Jaw for Cancer.*

A historical review of the first resection done by Gensoul, chief Surgeon of L'Hotel -Dieu of Lyon, May 26, 1827, is made. The patient, a young man named Vericel, living at St.-Laurent de Chamousset, near Lyons, was threatened by death due to a malignant cancer of the right side of the upper jaw and his case was considered hopeless. His trip to Lyon was his last hope for cure. The famous surgeon, whose courage was shown in a picture of the revolutionary days of 1833, when he alone stopped a violent mob from entering his hospital and murdering the patients, was hesitant to treat such a tumor. He would have refused operation if the patient had not threatened to commit suicide. After a long anatomical study of the pedicles of the jaw, he was ready to attempt the first resection. The patient was tied to a chair, without anesthesia, and the systematic resection was performed as quickly as possible, with the use of crude instruments. The patient recovered. Others, like Monique Tournier, were also operated on successfully for malignant tumors of the antrum.

Even after 126 years, although new methods of diagnosis and treatment are used, the principles of resection described in the "Surgical Letter" of 1833 remain the same.

The extension of the tumor is defined by a series of roentgenograms and the indications for total resection of the skeleton of the antrum are suggested. The operation is performed with the patient under anesthesia produced by intralaryngeal entubation and pharyngeal plugging with gauze. Each stage of the operation is explained by animated

cartoons, and the use of the proper instruments is taught.

Examination of tissue specimens obtained at operation corroborates the radiological data. Some results are presented showing today, as in the last century, the accuracy of Gensoul's operation.

L. DURAND: *Indications and Results of Ureterocystoneostomies in the Treatment of Renal Tuberculosis.*

The importance of pelvic and intramural lesions of the ureter in the development of renal tuberculosis and their more or less rapid mechanical impingement upon the upper adjacent kidney—which may be healthy or contain specific lesions—justify the employment of conservative ureteral surgery.

The antibiotics permit the employment in tuberculous ureteritis of methods that have been used for a long time in the treatment of other ureteral lesions, fistulas, or congenital malformations.

Many techniques are used for ureterocystoneostomies:

1. Side-to-side ureterovesical anastomosis. This operation requires a healthy bladder, a nongrowing ureteral lesion limited to the meatus or to the intramural ureter, and significant dilatation or distention of the pelvic ureter.

2. Ureterovesical reimplantation: Puigvert's operation. This may be accomplished in the following ways:

(*a*) By transvesical approach.

(*b*) By extravesical approach, permitting a wider dissection of the ureter and a more minute hemostasis, but one that leaves the pathologic segment in place, thereby running the danger of contaminating the pelvic cellular tissue.

(*c*) By combined extravesical and intravesical approach.

Puigvert's operation requires a healthy bladder, and the ureteral lesions cannot exceed 6 cm.

Tubular vesicoplasty: Boari-Casati's operation. Anastomosis between the ureter and the pediculated vesical fragment may be accomplished either by end-to-end anastomosis or by intubation. This operation permits the removal of 10 cm. of ureter.

Ureterocystoneostomies may be combined with the following operations: treatment of partial lumbar or pyeloureteral ureteritis; partial nephrectomy; cervical resection.

The advantages of these operations are manifold.

By eliminating obstruction and ureteral stasis, the mechanical effects of these conditions are removed, namely, the ureterohydronephrosis, which would sooner or later cause the functional death of the kidney.

By suppressing ureteropyelic stasis, they would permit and augment the effect of antibiotics on parenchymatous lesions.

They are not radical, and, in case of failure, nephrectomy or higher derivation of urine may be performed.

But they have some drawbacks; these procedures may be complicated in the following ways: fistulas, often transitory; hemorrhage requiring a second operation; infection, either pelvic cellulitis or pyelonephritis; vesicoureteral reflux which is frequent in the postoperative period but rare two months later. This latter complication would be less frequent after the operation of Boari than after the operation of Puigvert.

The indications for operation can be decided only after study of the condition of the ureter, the bladder, and the kidney. Hence the importance of intravenous urography, using time exposure for the negatives, cystography which alone can show reflux, and, as a supplementary procedure, upward pyelography when the results of urography are insufficient, and downward ureterography or iliac ureterography done preoperatively when roentgen studies of the kidney are unsatisfactory.

Despite all these tests, the state of the ureter cannot always be judged with certitude. There are pictures that give false impression of ureteritis. The meatitis and vesical intramuritis may be cured by medical treatment at the same time as the cystitis. Some pelvic ureterites of the ascending or descending types, are progressive; others are not.

Therefore, the operative indication for ureteritis can be estimated only after the progress of the lesions is known, after medical treatment, and after repeated roentgenologic examinations.

Once the diagnosis is determined, surgical treatment is justified either by the importance of functional symptoms (repeated acute pain in the kidney) or by the progressive lowering of the functional value of the functional renal value.

The indications are different in unilateral tuberculosis, and bilateral tuberculosis, and in the kidney that remains after nephrectomy.

In unilateral renal tuberculosis, ureterocystoneostomy is contrasted with the radical operations, nephrectomy, or nephrourectomy. It should be kept for cases in which the bladder is healthy and the parenchymatous lesions are of little importance. If the extension of the ureteral lesion is less than 6 cm., Puigvert's operation is the logical choice; if it is more than 6 cm., then Boari's operation becomes necessary.

If the parenchymatous lesions are located at one end, it is worthwhile to combine ureterocystoneostomy with partial nephrectomy; partial nephrectomy should be the first step. In case of failure, nephrectomy or nephrourectomy should be performed.

The possibility of conservative operations on the pelvic ureter has changed the criteria for surgical treatment in cases of bilateral tuberculosis. In some cases, tuberculous kidneys destroyed by progressive mechanical dilatation can be preserved, but the indications for Puigvert's or Boari's operation are rarer than in cases of unilateral tuberculosis, for vesical lesions are much frequenter.

The kidney remaining above a pelvic uteritis can be healthy or tuberculous. Indications for ureterocystoneostomies are exceptional for frequently ureteritis of vesical origin is associated with a small bladder. If the bladder is healthy, the kidney not tuberculous, stenosis limited to the meatus or to the intramural ureter, and the pelvic ureter very dilated, side-to-side ureterovesical anastomosis is certainly the best operation. If the ureteritis is more extended, Puigvert's or Boari's operation should not be considered. In case of failure, higher derivation of urine can be done.

The results may not be estimated definitely, for the time of response is not sufficient; it is not more than two years. In 13 cases without operative fatality, we had 4 failures, all due to bad diagnosis. In three cases, small vesical lesions continued to extend; in one case, pyeloureteral stenosis complicated the result. In all the other cases, the results were excellent, judged from all functional tests; the patients did not suffer further nephritic pains. Urographic studies made postoperatively all showed a disappearance of stasis. The results in cases of parenchymatous lesions is indefinite; we have discovered only that these lesions have not been aggravated.

These conservative operations on the pelvic ureter are of interest, but they are indicated in only certain limited cases.

J. CIBERT: *Ileocystoplasty in the Treatment of Chronic Tuberculous Cystitis which Remained Uncured by Nephrectomy (Twenty-Three Cases).*

Patients who have cystitis after nephrectomy done for tuberculosis need to be relieved from frequency of micturation and sometimes of incontinence. Furthermore, their lives are endangered by the destruction of the remaining kidney by ureteral changes caused by lesions of chronic cystitis (vesicoureteral reflux or ureteral stenosis of the lower section of the ureter).

In treatment of these patients, we can employ enervation of the bladder (which is ineffective against bladder retraction and reflux), ureterostomy, nephrostomy (which produces physical inabilities) and ureterosigmoidal implantation (generally followed by death within a few years).

Ileocystoplasty, an operation that is still seldom used, has remote results which are little known. But its favorable effect in cases of vesical disturbances and in those of renal alterations cause it to be considered as the best treatment for chronic cystitis.

P. SANTY: *Anatomic Forms of Stenoses of the Isthmus of the Aorta.*

This is an account of the anatomic findings in 60 operations for coarctation of the aorta performed since Oct. 30, 1948. Preoperative studies consisted of aortography done either by the venous route or by the retrograde route. These studies were made primarily to eliminate the forms of stenosis that cannot be treated by radical operation.

The majority of the patients who underwent operation were men (40 males and 20 females), and 40 of them were less than 21 years of age. The most frequently encountered anatomic conditions were the following:

20 cases: the substenotic segment was long, which permitted the accomplishment of Crafoord's operation under favorable conditions.

4 cases: the upper segment was long but narrow, and it was necessary to perform an extensive resection to provide the anastomosis with a sufficient diameter. In one of these cases, we used Clagett's technique.

23 cases: the upper segment was very short, compelling us to make the anastomosis partly on the aortic concavity, partly on the hypertrophied subclavian.

12 cases: there was no substenotic segment; the descending aorta was implanted by creating an almost total diaphragm on the convexity made by the subclavian and the aorta.

In six cases we had to create an orifice in the aortic convexity and to implant the descending aorta in this orifice. In one case, a Blalock anastomosis was made, causing cerebral injury. In this case the right subclavian began below the stenosis, an anomaly which we also observed in another case.

In only one case did we find the stenosis associated with an enormous arterial duct.

Among these 60 operations, 4 failures were encountered:

1 embolus and mesenteric thrombosis
1 hyperthermic shock
1 cerebral injury after a Blalock-type operation
1 high medullary ischemia after ligation of numerous collateral vessels.

At the present time all the intercostal vessels are preserved. In no case was the arterial graft used; we have always been able to perform the direct operation except in the two cases cited previously in which Clagett and Blalock's techniques have been employed.

All 56 survivors benefited by the operation and noted that the blood pressure in both arms and legs remained normal. No accident or later postoperative complication occurred.

P. MARION: *Approach and Techniques in Mitral Commissurotomy.*

Twenty-five years after the first attempts were made by Cutler, Levine, Beck, Allen, and Soutar, the use of surgical treatment for mitral stenosis was accepted, thanks to the work of Bailey, Brock, Harken, G. Murray, and Smithy.

1. The mitral valvulotomy approach

A. Thoracotomy

We prefer to employ the small axillary inframammary thoracotomy with partial resection of the fourth rib. By this approach it is possible to temporarily clamp the carotid vessels.

B. Cardiotomy of the atrium

First case: the auricle is flexible, permitting the passage of the index finger.

We have given up the use of hemostasis by clamp as we prefer the Rumel tourniquet, a trustworthy, precise, and practical method.

Second case: The auricle is not available (because of thrombosis and and retraction).

(*a*) When the thrombosis is limited to the auricle, the previous extraction of clot is indispensable to perform the commissurotomy by the auricular route. With this procedure the risks of embolism are considerable.

(*b*) When the operation is performed by using the pulmonary vein, commissurotomy is done and auricular thrombosis is avoided. This route is indicated when the thrombosis reaches the top or the entire right part of the atrium.

2. Technique of Mitral Commissurotomy

In its flexible form the great mitral valve, raised at each systole, sometimes hides the narrow oval orifice, which has a diameter of from 6 to 12 mm. at its large axis. The index finger can be inserted into the orifice and separate the commissures very easily. The new orifice is often 2 fingerbreadths in width.

According to our statistics, this flexible form is seen in about 44% of cases.

The annular fibrous forms are very different. The oval mitral orifice, reinforced by a thick, resistant, fibrous plaque, can be easily penetrated. But when the index finger is removed, the orifice again returns to its previous dimensions. Otherwise, the commissures may break at 2 or 3 mm., and the result is not satisfactory.

In the fibrous annular forms, instrumental commissurotomy is always necessary. This is equally true for the treatment of the valvular fibrous forms.

The suborificial funnel-shaped fibrous form is only a variety of the valvular fibrous form.

During the enlargement of a fibrous orifice, one locates it and sometimes it is possible to separate the chords that constitute the funnel. More frequently, the suborificial stenosis is caused by retraction of the cords or by hypertrophy of the pillars. In such a case, surgical treatment is useless. There is no parallel between the clinical stages and the anatomic progress of mitral stenosis. In our series, we had to perform instrumental commissurotomy in 45 of the last 127 cases.

The treatment of the calcified fibrous forms and the forms with insufficiency is discussed by the author. Our experience includes 187 commissurotomies performed by P. Santy and P. Marion.

M. Jaubert de Beaujeu: *Segmental Resection for Pulmonary Tuberculosis (One Hundred Seventy-Two Cases).*

From October, 1950, to February, 1954, 172 segmental resections were performed for tuberculosis, with the following types of operative procedure: One segment or a subsegment only (84 cases); several segments (88 cases).

The lesions treated in this way are irreversible ones with segmental localization, either as systematic caseous masses or irretractile cavities surrounded by thick caseofibrous shell or filled cavities.

Radical resection was done in 93 cases and focal resection in 79. Previous collapse creates special conditions for resection.

Technically, several points are important: The "traction-dissection" method to isolate the segment; control of venous hemostasis and air leaks; pleuralization of the bronchial stump; freedom of lung expansion; decortication; vigilant postoperative care; complementary collapse (in some cases).

Postoperative course has been uneventful in 83.7% of cases; complications occurred in 16.5% of cases, and one death occurred (0.6%).

The nontuberculous complications (nine cases) were cured, except in one case of air metabolism.

The tuberculous complications were cured in 8 of 10 cases; three patients are improving.

Phthisical results of the 110 first cases have been studied (October 1950 to May 1953) so as to have a follow-up report for at least a year. In 100 cases (91%), the results are good; in 4 cases (3.6%), incomplete, and in 5 (4.5%), bad.

The results depend on the type of lesion (a caseous mass or cavity), the type of resection (radical in 96.7% or focal in 83.3%), and the type of evolution. The functional results are highly satisfactory.

M. Berard: *Results of Surgery of Pulmonary Exeresis in Child. Data Discovered in Course of 123 Operations.*

The statistics of P. Santy and M. Berard, based on 123 operations, show:

Bronchiectasis and cysts	95 cases
Tuberculosis	13 cases
Benign and malignant tumors	6 cases
Chronic pulmonary suppuration	4 cases

The age of the patients extends from 2 months to 5 years. The operations include the following:

21 pneumonectomies
94 lobectomies
9 segmental resections

From the viewpoint of operative mortality, one may distinguish the following two distinct periods:

In the first (from 1937 to 1948), there were seven deaths out of 29 operations (25% immediate or late mortality).

In the second (since 1948), there were three deaths out of 94 operations (a rate scarcely higher than 3%).

Furthermore, these three deaths occurred in one case after pneumonectomy for tuberculosis (bronchial fistula) and in two cases after lobectomy for bilateral bronchiectasis.

At the present time, our statistics show the almost complete absence of postoperative complications in 94 cases in which surgery was performed. On the whole, surgical exeresis is a much easier operation in the child than in the adult.

From a functional viewpoint, the later results of lobectomy or segmental resection are perfect, but after pneumonectomy the dangers—particularly in the very young child, are those of scoliosis and thoracic deformity much more than that of respiratory insufficiency. Actually, there is a good chance of functional adaptation in the child after pneumonectomy. In 12 out of 13 cases in children, pulmonary exeresis for tuberculosis resulted in complete cure. The results of bronchiectasis are also good, 70 excellent results being obtained in 84 cases.

P. GALY: *The Thymomas.*

The statistics and the descriptions deal with removable tumors. The cases treated only by radiotherapy or diagnosed at biopsy during exploratory thoracotomy have been eliminated. The thymomas include 8% of the cases of mediastinal tumors that have been subjected to exeresis (15 of 150 patients). These are tumors of the anterior mediastinal space, mainly of the intermediary stage. They are lobulated, attached by a vascular pedicle from their upper pole. The following types can be distinguished:

The thymolipoma—benign, voluminous, of a fatty appearance (one case).

Thymic cysts. It is rare to encounter a single cyst; they are usually multiple or cysts with mural tumor (4 cases).

Filled tumors, usually encapsulated. In one case, such a tumor was discovered between the trachea and the big venous trunk.

The tendency to invade is weak, even in the histologically malignant cases. Local proliferations are not found.

Except for thymolipoma, which is exceptional because of the abundance of fatty tissue, these tumors are constituted by the association of two cellular types: small lymphocytic cells; great reticulated cells, called epithelial.

The two types coexist. The noting of the juxtaposition of the two varieties is indispensable for diagnosis (lymphoepithelioma). According to the predominance of one type or the other, the aspect is either that of a pseudoepithelial form or that of a lymphoid form. The presence of Hassal corpuscles is rare (5 of 14 cases).

The histologic or cytologic signs of malignancy are rare (3 out of 14 cases). Diagnosis is often difficult in lymphosarcoma, small-celled bronchial cancer, and certain undifferentiated tumors of the pleura.

Clinically, aside from the cases of voluminous tumors in children which may produce signs of compression and thoracic deformities, the functional course is frustrating.

Radiologically, tumors of the anterior mediastinal space are found in a position lower than classically described (3 out of 4 cases in children): 3 upper, 5 middle, 2 lower. The shadow is round, polycyclic, with clearly defined edges, and unilateral. A shell-shaped calcification is sometimes seen.

The most difficult preoperative diagnosis to make is in cases of vascular or pericardial malformations. Angiocardiography is sometimes necessary. We had two cases of association: in one with myasthenia, a radiosensitive malignant tumor. Such an association was more rare in our series than in those of others: one tumor among 14 patients with myasthenia; one tumor among 4 patients with myasthenia on whom operation was performed.

With leukemia: This case was an 11-year-old child, with a tumor that was clinically and histologically malignant, of the lymphosarcomatous type. The thymic nature is certain because of the connections. At the time of operation the hemogram and the myelogram were normal. Five months later, an acute case of leukemia supervened.

Malignancy and benignancy are variously appraised in the various statistics: all conditions are at times considered malignant and at times benign. Malignancy is perceived particularly locally and histologically. Clinical malignancy is often difficult to perceive.

An invading tumor of difficult exeresis and of malignant histological nature will have a long-time evolution that is favorable for survival. A cystic tumor, macroscopically benign although suspected of being histologically malignant, may occur after 11 years.

The operation was most frequently performed by the lateral route (3 sternotomies). Except for two deaths in the hospital (myasthenia and decompression disorders caused by an enormous tumor in a very young patient), one death from leukemia and one recurrence sensitive to radiotherapy after 11 years, the results have been favorable.

Radiosensitivity in the two cases studied was variable; it was nonexistent in a large thymoma in a child and clearly present in the case associated with myasthenia.

P. MICHAUD: *Treatment of the Megaesophagus by Heller's Operation.*

We have always preferred Heller's operation to cardioplasties and esogastrostomies. The latter, indeed, are much more serious and frequently provoke peptic esophagitis and esophageal ulcers. Heller's operation is much simpler, quicker and more benign; it is better suited to the benign nature of the disease and can be performed even on delicate patients (children, the aged, and cachetic persons). Our statistics include 146 cases.

1. The technique of Heller's operation must be perfect in order to obtain good results with it.
   The thoracic route is rarely used.
   The abdominal route is less dangerous and quite adequate (it can be enlarged by xiphoid resection).
   The liberation of the esophagus should be very extensive not only in the abdomen but in the mediastinum through the diaphragmatic hiatus. This procedure permits the section of the nerves reaching the esophagus. (This neurectomy has a definite functional result.)

Myotomy begun at the cardia must be carried very high up the thoracic esophagus (6 to 10 cm.). It should be completed, exposing the mucous membrane. The procedure is continued for an equally long section on the stomach.

The wound of the mucous membrane is practically the only danger in this operation; its suture, protected by an epiploplasty (and not by the closing of the myotomy), usually avoids fistulization. A drain for aspiration and a Levine tube for alimentation decrease the danger of fistulization when it does occur.

2. Results of Heller's operation

Mortality:

Five deaths in 146 cases.

Three wounds of the mucous membranes

2 peritonitis

1 mediastinitis

Morbidity: This consists only of esophageal fistula. We have seen seven cases, healed in 8-12 days by the use of aspiration (only one needed transpyloric gastrotomy).

Functional Results: These results are judged in the following ways:

1. The subjective evaluation of the patient
2. Gain in weight
3. Radiologic changes.

A. Total Results

Total	Lost from View	Failure	Fairly Good	Excellent
141	4	11	21	105

B. The results judged according to postoperative periods are very important for the objection to Heller's operation is that it is short-lasting in its effects. Our statistics show:

Period Postoperatively	Perfect Results
Less than 18 months	80%
18 mo.-2 yr.	76%
2 yr.-5 yr.	75%
5 yr.-10 yr.	78%
More than 10 yr.	77%

C. Study of results in respect to the age of the patients shows that the younger the patient the better and longer-lasting are the results.

D. When the disorders are of long duration (fixed lesions of the esophagus caused by stasis) the results are less satisfactory.

E. Enormous megadolichoesophagi often are followed by unfavorable reactions but ultimately by excellent results.

Total Results:

72% are completely cured.
18% still have some difficulties.
10% are failures or unsatisfactory results.

3. Nature, Cause and Treatment of Failures

A. Dysphasia is the most frequent disorder

This may be due to the following:

1. Incorrect diagnosis (esophageal or paraesophageal lesion, ulcer of the cardia, or plastic peritonitis).
2. A technical mistake. An iterative Heller operation might be necessary after the failure of medical treatments. It is more simple by the thoracic route which also permits a major operation (resection of the cardia rather than cardioplasty or esogastrotomy).
3. Cicatrization about the cardia of the operative hematoma; sometimes dilatations produce good results.

B. Complications apart from dysphasia

Gastric atony caused by vagal trauma regresses quite rapidly.

Gastroesophageal reflux has been observed in five cases. It has never caused peptic esophagitis. Certain technical maneuvers can prevent it.

AMERICAN COLLEGE OF SURGEONS *

A SECTIONAL meeting of the American College of Surgeons—the first in this country—was held at the Royal College of Surgeons of England on May 17-19. A full programme included panel discussions on massive gastrointestinal hæmorrhage, preoperative and postoperative care, hand surgery, and intestinal obstruction; symposia on cardiovascular surgery, gynæcology, and cancer; papers on many diverse subjects; and visits to hospitals. In addition a Moynihan lecture and a Hunterian lecture were given by two of the visitors.

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This peaceful invasion of Lincoln's Inn Fields enabled British surgeons to listen to American workers who are well known here by name. The meetings, some of which are briefly outlined below, were conducted in a refreshingly brisk and forthright style.

Massive Gastro-intestinal Hæmorrhage

Moderator: Prof. FRANK GLENN

The proceedings of this lunch-time meeting opened in earnest somewhere between the peach melba and the coffee. The panel were first allowed a little rein to speak in general terms on different aspects of the subject. In contrast to the teaching that persists in many textbooks, panel members insisted that hæmorrhage from acute ulcers, as well as from chronic ulcers, can be dangerous to life. All were in favour of the indwelling gastro-œsophageal tube in the management of massive hæmorrhage from the stomach, as a means of indicating the earliest moment at which hæmorrhage recurs after having ceased.

Prof. WALTMAN WALTERS (Rochester, Minnesota) pointed out the need for a thorough search of the whole intestine when operation was undertaken for hæmorrhage, where there was no obvious lesion in the stomach and duodenum.

Mr. NORMAN TANNER expressed the view that œsophageal varices bleed as the result of ulceration of their surface, rather than from traumatic rupture. He had had 41 cases in the last ten years: up to 1947 two-thirds of the patients had died, but since then the mortality had been reduced to one-third with more vigorous treatment. This consisted in two stages: (1) immediate arrest of the hæmorrhage by traction on an intragastric balloon which was distended by means of an œsophageal catheter; and (2) if hæmorrhage recurred on removal of the balloon after a time-limit of two days, division of all veins round the cardia followed by subcardil transection and re-suture of the stomach, whereby the œsophageal varices were completely separated from the portal system. Most cases of gastro-intestinal hæmorrhage were due to a gastric or duodenal lesion, and unless the diagnosis was obvious from the history or signs it was his practice to gastroscope the patient, since a bleeding lesion in the stomach or indeed in the œsophagus could be more easily seen by the gastroscope than by any other method.

Prof. C. F. W. ILLINGWORTH thought that the massive hæmorrhage from a submucosal vessel might possibly be due to the actual rupture of the vessel as a primary lesion and might not necessarily be due to an acute erosion. He complained that, whereas formerly he had had difficulty in getting the cases from physicians early enough for operation, it was now difficult to hold them back from surgery, and he felt that they ought to learn to treat their patients medically—by which he meant the use of the indwelling aspiration tube. When the cause of the bleeding could not be ascertained at operation, even after opening the stomach for inspection, he was in favour of blind gastrectomy on the ground that 70% of acute erosions were in the pyloric half of the stomach.

Mr. IVOR LEWIS recoiled from the advocacy of blind gastrectomy, holding that the lesion must be identified if necessary by gastrotomy. In the case of duodenal ulcer it was his practice to open the duodenum, underrun the bleeding-point, and proceed to partial gastrectomy, or, if this was not possible, to gastro-enterostomy. He warned against leaving the bleeding artery in the duodenal stump untied merely because it appeared to be sufficiently occluded by clot. In his experience of 2 patients in whom ligation had been considered unnecessary for this reason, I had died from recurrent hæmorrhage and the other had barely escaped with his life.

After these preliminary skirmishes came the real encounter—question and answer by the panel.

By what criteria was "massive" hæmorrhage held to justify operation?

Professor WALTERS: If the patient is older than 45 years, especially if he has had pain or a known ulcer, and particularly if the hæmorrhage has occurred.

Professor ILLINGWORTH: Never operate at first hæmorrhage. Patients do not die from it if they have got to hospital. Operate only if the bleeding continues as indicated by the indwelling tube.

Mr. TANNER: At St. James's Hospital 55% of the cases were aged over 60; so on the whole they were suitable for surgery. If surgery seemed to be indicated anyhow, he would operate even after a single severe hæmorrhage. The rest were gastroscoped and the site of the bleeding lesion was identified. Operation was undertaken on these only in the event of recurrence of the hæmorrhage.

Mr. LEWIS emphasized the great value of the indwelling tube, which enabled operation to be undertaken before a rising pulse-rate indicated a general collapse.

What do you do if you operate for massive hæmorrhage and fail to find the bleeding-point?

Professor ILLINGWORTH: Do a blind gastrectomy.

Mr. TANNER did not often find himself in this position because he gastroscoped the patients first, but he would do a blind gastrectomy.

Mr. LEWIS would never do a blind gastrectomy. Why, the next step would be for someone to report doing a blind gastrectomy because he could not find a perforation!

Professor WALTERS would do a blind gastrectomy because severe bleeding could come from multiple

acute erosions which could be neither seen nor felt at operation, but which could be seen afterwards in the specimen under the microscope. He mentioned work in the U.S.A. which indicated that in fact bleeding seldom recurred after blind gastrectomy in these circumstances.

Is it sufficient to underrun the bleeding vessel and not resect the stomach?

Professor ILLINGWORTH: No.

Mr. TANNER: No, except for stomal ulcers. In 10 duodenal cases he had simply underrun the bleeding vessel and added a gastro-enterostomy. 2 bled again; 1 died.

Mr. LEWIS: No, except in some cases of duodenal ulcer.

Professor WALTERS: No, except in some cases of duodenal ulcer when it is unsafe to do a partial gastrectomy, but gastro-enterostomy should be done instead.

Would you resect the stomach for a bleeding ulcer in a child of 15?

Mr. TANNER: Yes; but he had no experience of such cases.

Mr. LEWIS: No experience, but he had done this in an 18-year-old patient.

Professor GLENN: Do a partial gastrectomy.

Professor ILLINGWORTH: No experience.

Is radiology useful to find the site of a bleeding ulcer?

Mr. LEWIS: No, because you are no better off if the result is negative.

Professor WALTERS: Yes, if carefully used.

Professor ILLINGWORTH: Yes, to exclude varices.

Can massive hæmorrhage occur from stomal ulceration?

Professor WALTERS: Yes.

Professor ILLINGWORTH: Yes, but not usually dangerous.

Mr. TANNER: Yes, but rarely really massive.

Mr. LEWIS: Yes, but usually only from acute ulcers.

If the massive hæmorrhage is found at operation to be due to œsophageal varices with portal hypertension, is immediate portacaval anastomosis indicated?

Mr. TANNER: No, porta-azygos disconnection (by gastric transection) is better.

Mr. LEWIS: No. He would temporise by the compression balloon.

Professor ILLINGWORTH: No experience.

Professor GLENN would control bleeding by traction bag, close the abdomen, resuscitate the patient, and prepare for a portacaval shunt if the hæmorrhage recurred when the bag was released.

Can massive bleeding arise from diverticulitis in the colon?

Professor ILLINGWORTH: No experience.

Mr. TANNER: No experience.

Professor WALTERS: No experience.

Mr. LEWIS had treated one case successfully by resection of the colon.

Has vagotomy any place in the treatment of massive bleeding from duodenal ulcer?

Mr. TANNER: No. Only useful afterwards.

Mr. LEWIS: No.

Professor ILLINGWORTH: No.

Professor WALTERS: No.

Are local agents, instilled through an indwelling gastric tube, of use to control hæmorrhage from the stomach?

Mr. LEWIS had used adrenaline but was sceptical of its value and had given it up.

Professor WALTERS: No.

Professor ILLINGWORTH: No.

Mr. TANNER: No.

What lesions of the small gut can cause massive hæmorrhage?

Professor WALTERS: Do not forget to search the second and third parts of the duodenum for a tumour—usually leiomyosarcoma.

Have you ever resected the stomach for massive hæmorrhage from a Curling's ulcer after burns or trauma?

Professor WALTERS: No experience.

Professor ILLINGWORTH: No experience.

Mr. TANNER: No, but one patient had died from hæmatemesis after burns. Necropsy had shown a gastric ulcer.

Professor GLENN had seen one patient bleed to death on the tenth day after a burn. This patient should have been operated on.

This good-humoured cut-and-thrust was strictly refereed by Professor Glenn, who made the panel stick to the point of the question. It was an agreeable change from the usual formal discussion of a technical subject.

Preoperative and Postoperative Care

Moderator: Prof. WALTER G. MADDOCK

Prof. F. A. R. STAMMERS, commenting on the importance of fluid and crystalloid balance, contrasted the resilience of the young, healthy patient with the sensitiveness of the elderly, who withstand disturbances of fluid balance poorly. Particularly in this group it is important to restore "physiological balance" before operation. Professor Stammers declared that the treatment of fluid and electrolyte disturbances is the responsibility of the surgical team, and emphasized the size of the deficits that

may develop in conditions such as intestinal obstruction, and the value of clinical examination and simple urine tests in the management of these cases. He recommended a daily intake of 2.5 litres of water containing 8-12 g. of sodium chloride and 2-3 g. of potassium chloride, and indicated the dangers of water intoxication from indiscriminate administration of fluid subcutaneously or rectally. Finally he referred to potassium deficiency and the need for accurately charting the patient's intake and output.

Prof. J. Garrott Allen (Chicago) discussed the problem of a "man-made disease"—homologous-serum jaundice. In a large series he found that this condition occurred once in every 200 blood-transfusions, but 20-50 times in every 200 transfusions of pooled plasma; and the mortality-rate of those affected varied, in different series, from 5 to 28%. In Professor Allen's view pooled plasma should no longer be used, but should be replaced by liquid plasma stored at room-temperature; this never gave rise to jaundice. Viruses could survive long in dried or lyophilised plasma, but only for a few days in cell-free plasma, which was stable and remained fit for use for at least three years. Plasma alone did not suffice for anæmic patients, who also needed blood-transfusions.

Prof. Macleod Douglas discussed the prevention of abdominal-incisional herniæ in the obese. He contended that the main strength of an incision derived from its fascial, aponeurotic layers, and that these should always be sutured with fine, interrupted, non-absorbable sutures. He described experiments showing that healing in fascial tissue takes some ninety days, and that until that time the tensile strength of the wound depends largely on the sutures. If possible self-opposing incisions should be made, and in general transverse abdominal incisions were preferable to paramedian.

Professor Maddock explained that the source of the gas causing postoperative abdominal distention, or "wind," was swallowed air. Apparently many patients become aerophagists for a few days after an operation; even if these patients belched frequently they never returned as much air as they swallowed. Professor Maddock described experiments showing that swallowed air was passed down the alimentary tract very rapidly, reaching the cæcum in ten minutes and the rectum in twenty-five to thirty minutes. He strongly recommended prophylactic continuous gastric suction after major abdominal operations, to prevent the development of "wind," and insisted that the abdomen should never be closed over distended intestines, which should first be emptied by aspiration.

Hand Surgery

Moderator: Prof. Michael L. Mason

Introducing this panel discussion, Professor Mason spoke of the great increase in the scope of hand surgery. Not many years ago it was concerned mainly with infections, whereas now interest was centered on reconstruction after injury.

Mr. Patrick Clarkson first referred to the common minor injuries of the hand which are treated in casualty departments. He described the arrangements at Guy's Hospital, and said that although special accommodation and equipment were necessary these were of a simple nature. He emphasized the importance of teaching the principles of traumatic surgery on these cases and of the planned delegation of responsibility.

Dr. Georges E. Cloutier (Montreal) described the primary repair of hand and finger wounds, and also spoke of the importance of proper facilities and technique. Complete debridement of the wound was more important than the administration of antibiotics. Conversion of the open wound to a closed wound by suturing or grafting was an essential part of the primary treatment. Full-thickness skin-grafts gave the best results, but he though that split grafts were safer with contaminated wounds. For pedicled flaps he sometimes used the side of the neck, while the local flap which he favoured was the cross-finger flap. When skin was avulsed it could sometimes be replaced as a full-thickness graft after careful removal of the fat.

Mr. W. C. Gissane dealt with closed fractures of the hands, pointing out that the orthodox conservative treatment of these often gave disappointing results. He described the open reduction of some common fractures and dislocations, saying that repair of the associated damage of the soft tissues was sometimes all that was required to make reduction of fractures stable.

Mr. R. G. Pulvertaft agreed with Mr. Gissane about the repair of fractures, and went on to speak about the treatment of divided tendons, discussing the indications for primary and secondary repair. Repair of the flexor tendons in the middle and proximal segments of the finger, where grafting was often necessary, was best deferred. At the wrist, and usually also at the distal part of the finger, tendons should be repaired at the primary operation. When function was limited after tendon repairs tendolysis might be useful.

The panel were not in full agreement about the palmar flap for covering skin tips, but Mr. Clarkson made the point that replacement of lost tissue should be by tissue resembling it as closely as possible; he claimed that the palmar skin was the best from this point of view, and it also gave good sensation. The panel also agreed that if several fingers were completely "degloved" it was probably a mistake to try to save them all, and the less useful might be amputated.

With regard to fracture fixation, Mr. Gissane said that for the juxta-articular fractures soft-

tissue suture was usually adequate. For fractures of the shafts of the long bones of the hand he had found mattress sutures of wire satisfactory.

The panel seemed to agree that ischæmia produced by a pneumatic tourniquet, such as a sphygmomanometer cuff, could be safely maintained for two to three hours. It was suggested, however, that prolonged ischæmia might favour the development of wound infection.

Acute Intestinal Obstruction

Moderator: Mr. A. Dickson Wright

Public discussion of the most serious subject usually gains from a leavening of humour; but there is always a critical level beyond which a debate cannot stray without lessening its usefulness. Under the direction of a moderator introduced by Prof. Waltman Walters, of the Mayo Clinic, as "a well-known surgeon and raconteur," the panel opted squarely for entertainment.

Nevertheless some valuable points emerged. The three collaborators agreed with the modern view that suction with a long intestinal tube is of benefit in the treatment of certain cases, and that fluid and electrolytic balance are all-important. Regarding the treatment of large-bowel obstructions Prof. P. H. T. Thorlakson (Winnipeg) energetically defended cæcostomy in certain cases, though this view was attacked by Mr. J. B. Oldham; while Prof. M. John Waugh (Rochester, Minnesota) considered that in left-sided colonic obstruction due to a growth, colostomy 5-6 in. above the growth was a good operation, allowing the later excision of both growth and colostomy with end-to-end anastomosis.

Cardiovascular Surgery

Chairman: Prof. Henry W. Cave

In this symposium experts from both sides of the Atlantic gave short papers covering a wide field. Those accustomed only to the British way of conducting similar meetings may have regretted the absence of general discussion of the papers which a less crowded agenda would have permitted. They were reminded, however, that the sectional meetings of the American college serve as refresher courses for many American surgeons working in isolation from the great centres of surgical research—a fact that justifies their rather didactic character.

Mr. R. C. Brock's exposition of modern techniques in intracardiac surgery invested the relief of the various valvular stenoses with a deceptive appearance of simplicity. Simpler it has indeed become with the discovery that the chambers of the heart can be directly entered with instruments, thus rendering superfluous the retrograde introduction of valvulotomies through an artery—the means by which Brock first undertook the relief of an aortic stenosis less than ten years ago. How little cardiac surgeons are inclined to rest on their achievements was shown by Mr. Brock in referring to the method he has used of open operation on the valves and intercentricular septum under direct vision. A dry heart is secured by clamping the great vessels. This has been made possible by the introduction of hypothermia. The oxygen requirements of vital organs are thus reduced and the dangers from ischæmia of their tissues minimised. This illuminating glimpse of the intracardiac surgery of the future suggested that an extracorcorporeal circulation, with its enormously difficult management, may become superfluous.

The treatment of aneurysms of the aorta by resection and replacement by frozen arterial homografts is now a well-established procedure, as was shown by Prof. Michael E. DeBakey (Houston), who reviewed some 60 cases in which this operation was done. Until very recently its scope was limited to aneurysms which could be dealt with without interrupting the blood-flow through arteries supplying vital organs, such as the renal arteries and the cœliac axis. This limitation has now been practically overcome by operating under hypothermia, and these arteries have been clamped long enough for grafts to be inserted, without impairing renal or hepatic function. Only the spinal cord remains intolerant of the ischæmia resulting from the necessary section of numerous intercostal arteries in certain cases.

The management of arteriovenous aneurysms was discussed in a thoughtful paper by Sir James Learmonth.

Technical problems relating to the collection, preservation, and insertion of homografts have lately tended to dominate discussion of surgery of the aorta and of the peripheral arteries. It was significant that Prof. C. G. Rob, with experience of over 60 cases, dwelt rather on the clinical indications for reconstruction of the large arteries. He usefully reminded his audience that what is technically possible is not necessarily therapeutically advisable, and that below the level of the iliac arteries the replacement of segmental obliterations in arteriosclerotic patients is very apt to fail, either through local thrombosis or from subsequent crippling angina pectoris or fatal cerebral or coronary thrombosis. In these circumstances he regarded grafting as usually justifiable only in cases with established or threatening gangrene in which a major amputation was the alternative. This might be postponed for a year or more by a reconstructive operation.

The clinical aspect was also emphasized by Prof. Michael Boyd, who indicated the importance of classifying claudicants according to the severity of their functional incapacity. He regarded sympathectomy as the sheet-anchor in treatment, but in his view this was not indicated in the mild cases

which almost any conservative treatment would relieve, nor in the severest cases for which tenotomy of the tendo Achillis provided the surest and simplest relief. He regarded long-continued administration of large doses of alpha-tocopherol as a valuable means of prolonging the effects of sympathectomy.

Arterial spasm remains the dark horse of peripheral-arterial disorders. Its rôle in the causation of symptoms, and the efficacy of sympathectomy in combating it, has always been regarded with more reserve in this country than on the continent of Europe. Sir JAMES PATERSON ROSS said that the time had come to review what we know, rather than what we suspect, about the origin of arterial diseases. Sympathectomy, which is of great value in acrocyanosis with spasm of the arterioles, was useless in the treatment of spasm of the larger arteries such as occurred after trauma. He showed graphically by means of a film made by Mr. J. B. Kinmonth how this spasm is abolished by the local application of papaverine-sulphate solution.

Dr. FREDERICK W. COOPER, jun. (Emory, Georgia), also speaking of vascular spasm, wisely hinted that sympathectomy might benefit some patients even if no actual spasm existed, for in certain conditions abolition of normal vascular tone would improve the circulation in a limb.

Those who heard the symposium were left with the sobering impression that, while the seemingly impossible is being achieved again and again, therapeutic judgment remains the most necessary and the most difficult task of the cardiovascular surgeon.

Pancreatitis

In a Moynihan lecture Prof. WALTER C. MAC KENZIE (Edmonton, Alberta) observed that with increasing awareness of pancreatitis this disease was being diagnosed ever more frequently, particularly since it had been recognised that the lesion might vary in severity from mild œdema to fulminating necrosis of the gland. There seemed to be no doubt that in acute pancreatitis the essential pathological feature was the escape from the lumen of the gland of activated pancreatic juice; but it was far from clear why this occurred.

Professor MacKenzie described a case of acute pancreatitis following embolism of the cœliac artery and reported work showing that pancreatitis could be produced experimentally by intra-arterial injection of fat emulsions after ligation of the pancreatic duct. In some cases of pancreatitis evidence of intravascular fat could be found, and he suggested that this might, under certain circumstances, derive from the liver. Workers in Wangensteen's laboratory had noted the similarity between the microscopic picture of pancreatitis and the Schwartzman phenomenon—both gave rise to venous and capillary thrombosis—and they had produced pancreatitis experimentally by appropriately administered doses of bacterial toxins. Professor MacKenzie suggested that, while there were probably several ætiological agents in pancreatitis, in many cases the essential factor was the development of a vascular lesion while the gland was under a pronounced secretory stimulus.

Of the 97 cases of acute pancreatitis that he had treated, Professor MacKenzie classified 15% as acute œdematous lesions and only 6% as fulminating. In this series over 50% of the women, compared with only 25% of the men, had concomitant biliary disease, and the really severe forms of the disease were commoner in those with no disease of the gall-bladder or bile-ducts. Of the 5 patients who died 3 were in their thirties. In all cases there was a distinct rise in the serum-amylase level, but this was often transient and lasted only twenty-four hours. In a large series of control cases of other acute abdominal conditions a raised serum-amylase level was found only very rarely. The diagnosis of acute pancreatitis depended largely on awareness of the possibility of the condition supported by a raised amylase level.

Professor MacKenzie recommended conservative, nonoperative treatment. The blood volume must be restored by the infusion of plasma or blood, and it was probably in this way that albumin was beneficial. A careful check must be kept on the electrolyte balance, and acute disturbances of carbohydrate metabolism detected quickly. Every effort should be made to prevent stimulation of pancreatic secretion; continuous gastric aspiration should be carried out; fluids should be administered intravenously; atropine and 'Probanthine' should be given; and pethidine rather than morphine should be used to control pain. 45 cases had been treated in this manner, with a mortality of 9%.

If gall-stones or other biliary disease was present, this should be treated appropriately as soon as the acute attack of pancreatitis had settled down. Despite this treatment some patients developed recurrent pancreatitis; in 2 such cases sphincterotomy gave relief; but Professor MacKenzie had not found this operation of benefit in patients without gall-stones, and he suggested that in such cases bilateral thoracolumbar sympathectomy might be necessary to relieve pain. In the group of patients with chronic, as opposed to recurrent pancreatitis Professor MacKenzie had treated 4 presenting with obstructive jaundice. Even at operation it had not always been possible to be certain that the pancreas was not involved by a growth, but all 4 cases had done well following cholecystojejunostomy. A further 12 cases of chronic pancreatitis with coincident gall-stones had done well after cholecystectomy, but in certain selected cases with crippling pain excision of the pancreas might be necessary.

Progressive Exophthalmos

Prof. Howard C. Naffziger (San Francisco), in a Hunterian lecture on progressive exophthalmos, described how he first became interested in this subject. Twenty-four years ago a patient was led into his clinic, totally blind with severe exophthalmos and protruding red masses of conjunctiva. This patient had undergone thyroidectomy for Graves's disease six months before; six weeks later he died from intracranial infection, which was the usual outcome of the untreated disease. When, shortly afterwards, Professor Naffziger saw a nurse with a similar condition, rapidly going blind with progressive exophthalmos and choked discs, he did his first orbital decompression. Entering the anterior fossa by the lateral route he uprooted the orbit and removed as much bone as possible from the walls. The annulus of Zinn was cut to release the pressure on the nerve (but this had not subsequently been necessary). The operation had been altered latterly only in that the bony removal had been made more extensive. The ethmoids were not entered because of the risk of infection; but it is possible to encroach on the frontal sinus as Poppen had suggested and thus get more room.

The main feature of the disease was the greatly increased retrobulbar pressure, and for this reason orbitonometric measurements—i.e., measuring the tension in the orbit—were of more value than exophthalmometer readings, which measured the amount of protrusion of the eyeball. His most surprising statement was that there was no fat to be seen in these orbits at operation, but that the muscles were enormous, and that when the disease had been present for some time, these might show hyaline degeneration. Almost all the reported necropsy accounts of orbital dissections in exophthalmos had emphasised their increased fat content, and the work of Rundle, Pochin, and others suggested this as a cause of the exophthalmos. When the exophthalmos was progressive, increase of fat was clearly not the exciting factor; and this might possibly be another pointer differentiating ingravescent exophthalmos from the protrusion usually seen in Graves's disease.

Professor Naffziger had operated on only 40 patients in the years 1930-50 despite the fact that his hospitals were responsible for some 250 thyroidectomies a year. The condition was extremely rare, and the only indications for this operation were a threat to the eye or to vision. 65% of his patients were females and 29 had required bilateral operations. He had not succeeded in controlling the condition with any form of endocrine therapy or X rays.

As regards investigation of the ætiology of exophthalmos, the most profitable line seemed to be that pursued by Dobyns, of Cleveland, who had isolated from the anterior pituitary an exophthalmos-producing factor distinct from the thyroid-stimulating hormone.

Retrolental Fibroplasia

The demonstrations included one of experimental work on retrolental fibroplasia by Dr. Norman Ashton in the department of pathology of the Institute of Ophthalmology.

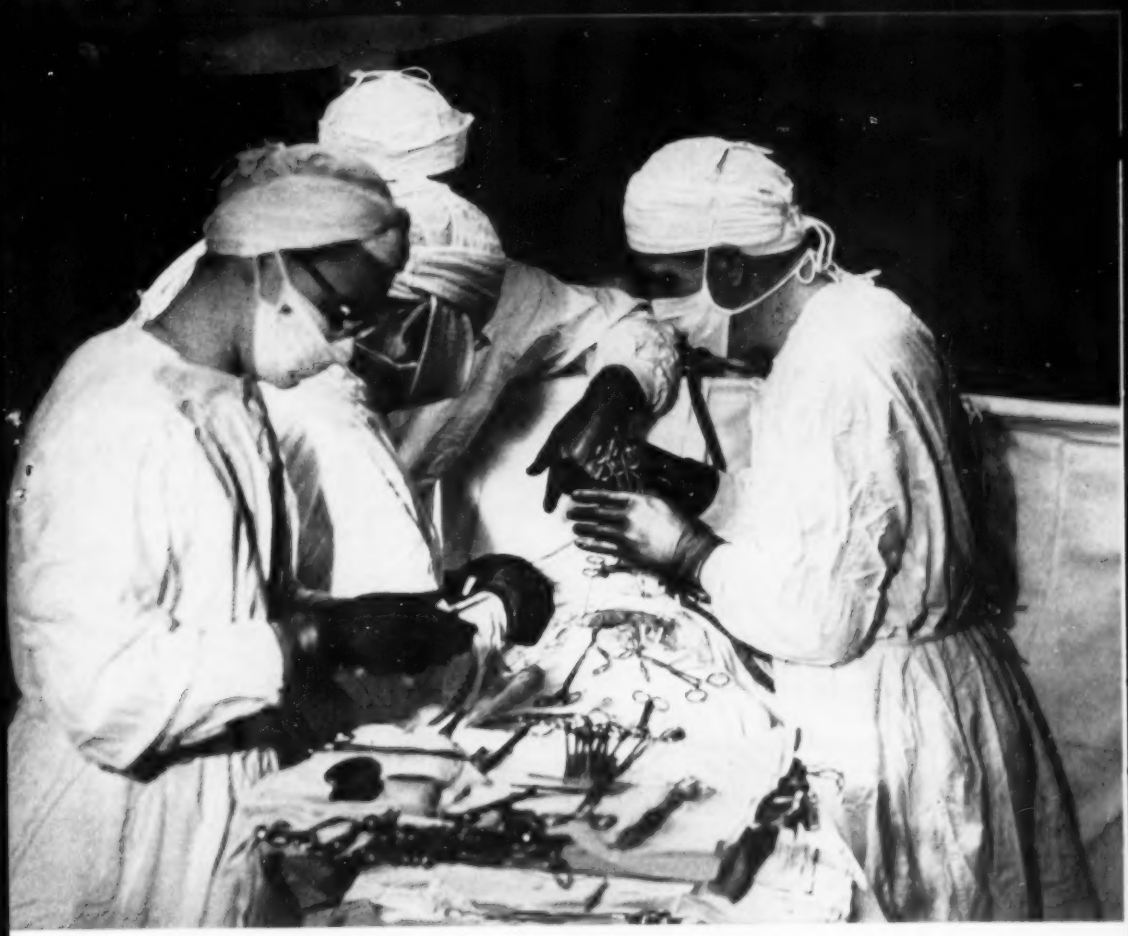
It was shown that high concentrations of oxygen at atmospheric pressure induce in the growing retinal vessels of the kitten a peculiar type of vasoconstriction which progresses to complete obliteration of the vessels, whereas concentrations below 35% have no such effect. This vascular closure is rendered permanent by thrombosis or adherence of the opposing vascular walls, so on transferring the animal to air severe anoxia develops in the retina and extensive and abnormal proliferation of vessels occurs, producing a picture which exactly parallels the early stages of retrolental fibroplasia in the premature baby. The two conditions thus appear identical in origin, and Dr. Ashton described some recent experiments which have further elucidated the problems involved.

It has been found, for instance, that the administration of ethyl biscoumacetate ('Tromexan') prevents intravascular coagulation within the obliterated vessels, which reopen normally in air—abnormal vasoproliferation does not then occur. While it is now clear that oxygen should be administered to the premature baby only when essential, and then only in the smallest quantity consistent with survival, it would appear that anticoagulants, such as heparin, may be the best preventive measure where the risk of exposure to high concentrations of oxygen must be taken.

Dr. Ashton and Mr. C. Cook demonstrated a new technique which permits microscopical examination of the living retinal vessels through a "limbal window"; by this means the visitors saw growing retinal vessels in a kitten's eye gradually and finally obliterated when the animal was subjected to high concentrations of oxygen.

Reports from Paris on Special Meeting of the International Society of Surgery in Paris and Strasbourg will appear in a forthcoming issue of the A. M. A. Archives of Surgery.

Editor.



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1. Parsons, W. H., et al.: Ann. Surg. 135:791 (June) 1952. 2. Whiting, J. A., and Hotz, R.: Surg., Gynec. & Obst. 97:709 (Dec.) 1953. 3. Beling, C. A., et al.: Geriatrics 7:179 (May-June) 1952. 4. Barbour, C. M., Jr., and Tennant, R.: J. Urol. 71:497 (April) 1954.

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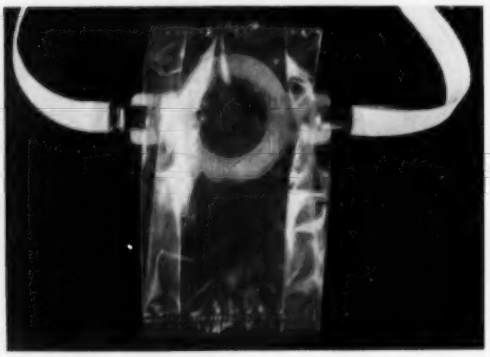

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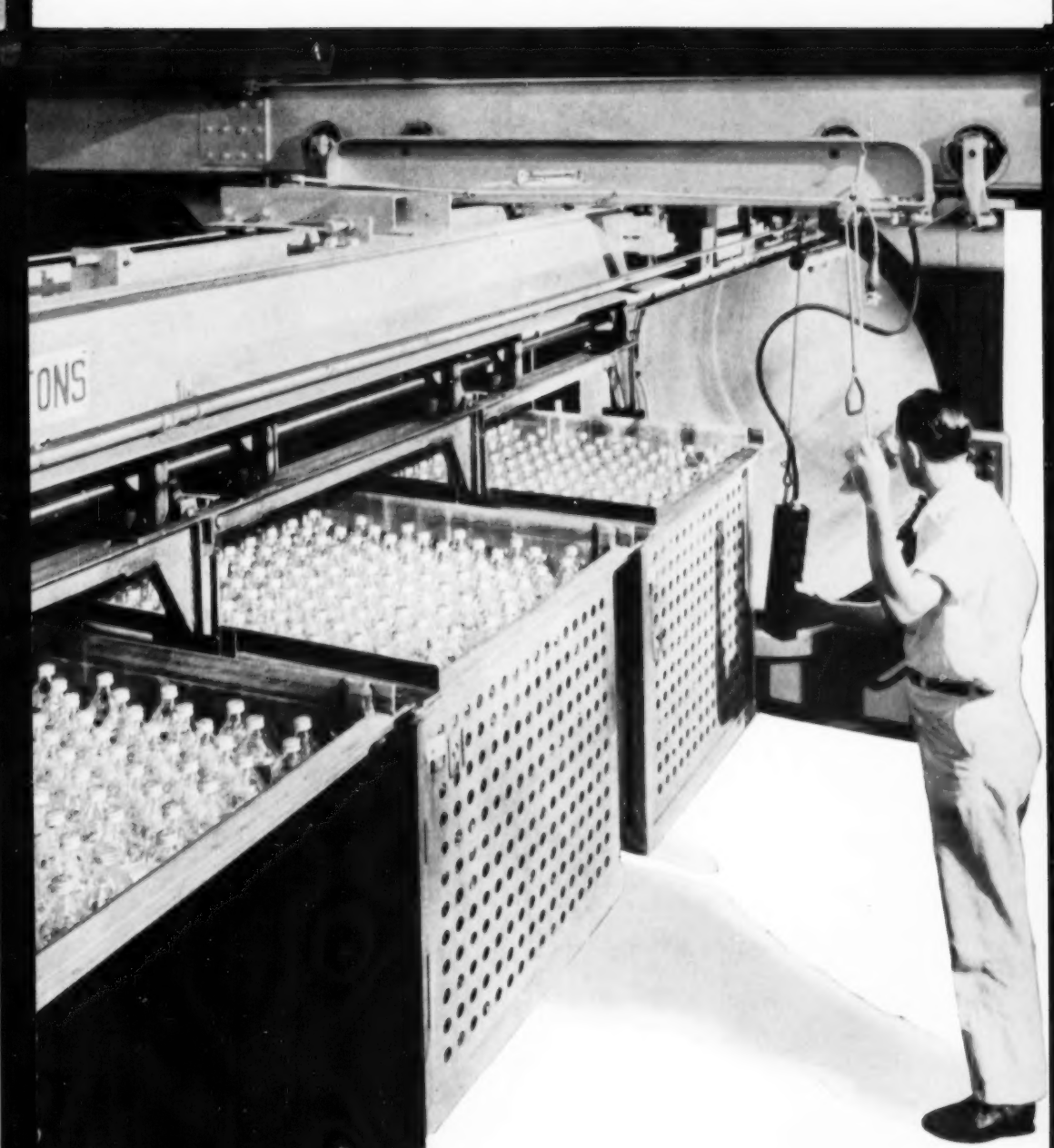

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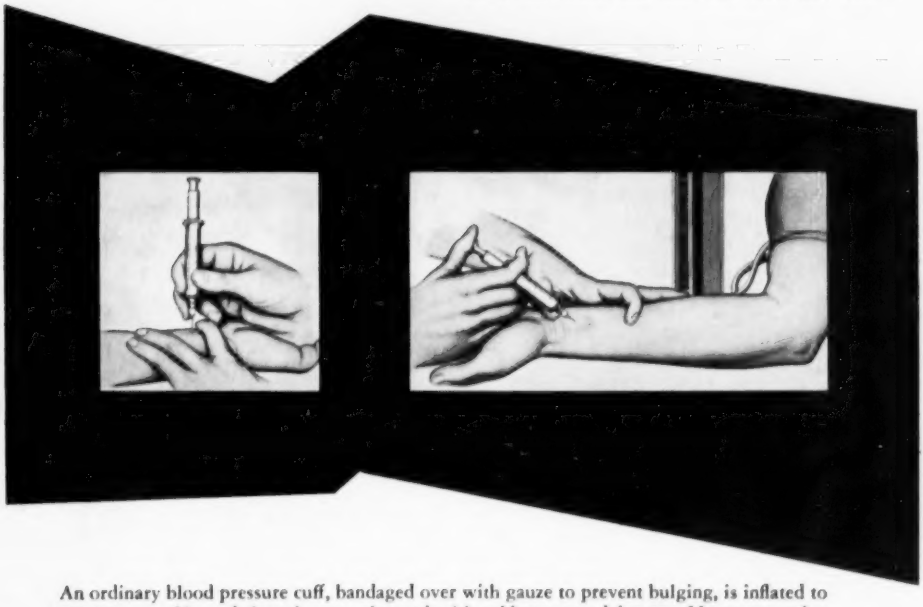

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